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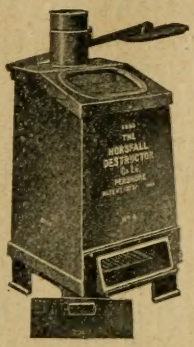

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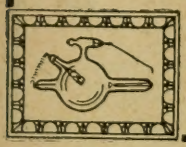

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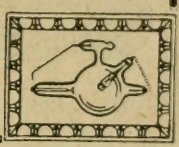

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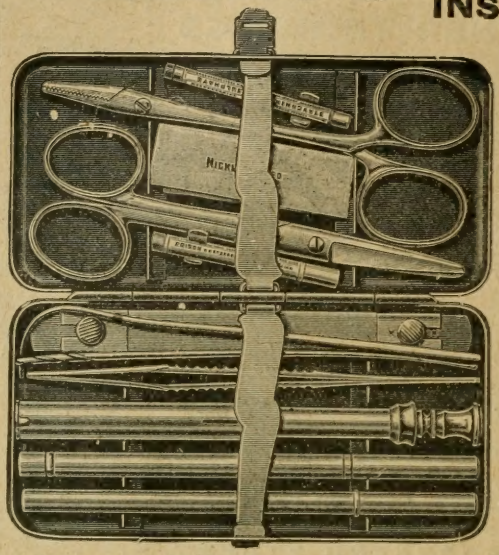

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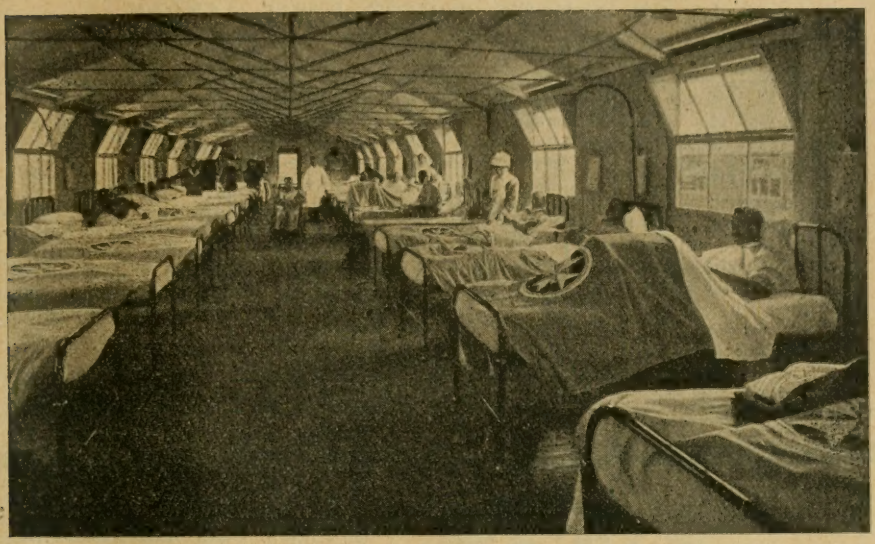

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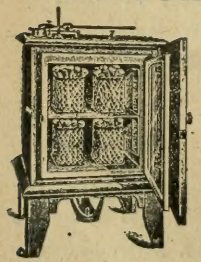

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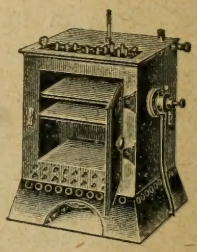

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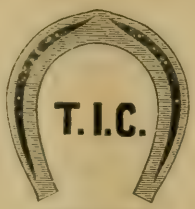

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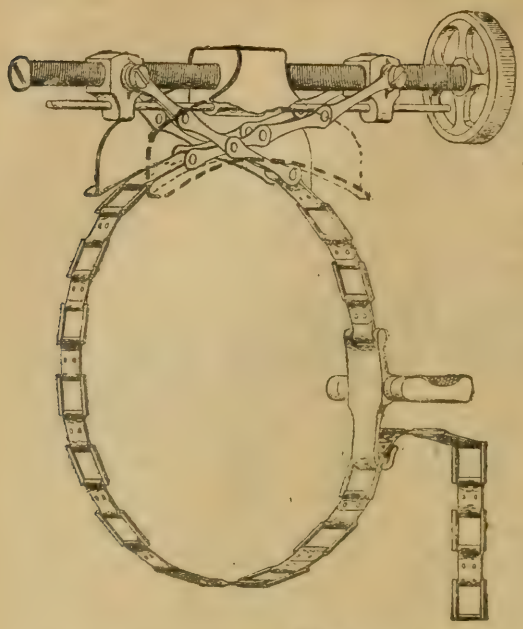

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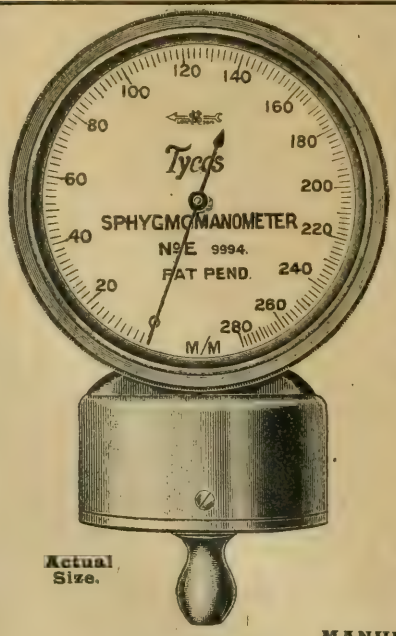

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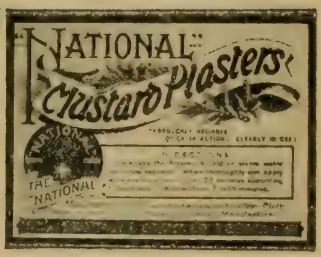

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ON THE PATHOLOGY AND TREATMENT OF POISONING BY TRINITROTOLUENE ("T.N.T."), AND ON THE ACTION OF CERTAIN OTHER POISONS WHICH AFFECT FAT METABOLISM.

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(*From the Hygiene Department, Royal Army Medical College.*)

CERTAIN obvious clinical and pathological features of cases of poisoning by trinitrotoluene, or trotyl, suggest at once their inclusion in a group to which may be referred also, (1) the cases of poisoning which occurred formerly amongst workers with tetrachlorethane in aircraft factories and cases of poisoning by dinitrobenzene ("D.N.B.") amongst munition workers, (2) cases of "delayed" chloroform and ether poisoning, after the administration of either for anæsthetic purposes, and (3) cases of acute phosphorus poisoning.

In all these cases there may be an acute manifestation of jaundice, sometimes with concomitant glycosuria, or acetonuria, as indications of disorder of metabolism. In all there may be found after death either fatty changes in the liver with some enlargement of the gland, or fatty changes in association with disorganization of the parenchyma cells, extensive interstitial fibrosis, and shrinkage of the gland as a whole. It has been presumed commonly that the two sets of changes represent earlier and later stages of a continuous pathological process.

Fatty changes have been described as occurring in the kidneys also.

more particularly in "delayed" chloroform poisoning, and in poisonings by tetrachlorethane, by dinitrobenzene, and by trinitrotoluene. An intense fatty engorgement of the liver, with some fatty engorgement of the kidneys, was found in the one case of "delayed" ether poisoning which the writer has had the opportunity of examining. "Fatty degeneration" of the heart muscle has been described, usually somewhat indefinitely, as occurring in some of these cases.

It is possible that yet another class of cases of poisoning, chronic arsenical poisoning, belong properly to this group. "Toxic jaundice" has occurred amongst industrial workers who are liable to inhale arseniuretted hydrogen. Similar jaundice has been recorded as occurring amongst French and German balloonists, as the result of inhaling hydrogen mixed with arseniuretted hydrogen. It has been assumed that the jaundice that occurs in these cases is hæmatogenous; but apparently there is not any available account of the exact changes found after death. It is known that fatty changes are of common occurrence in the liver in arsenical poisoning caused in other ways. Poisoning by arseno-benzene compounds comes perhaps within a category somewhat different from that of poisoning by mineral arsenical salts. It would appear that the toxic effects of certain organic arsenic compounds may be influenced materially by the benzene constituent. Experimental work in connexion with the special toxicity of arseno-benzene compounds has been in progress in the Hygiene Department of the College for some time past; and the results obtained so far appear to bring these compounds into close relation with the class of poisons now being considered.

It has been supposed generally in cases of poisoning by the group of six substances more particularly referred to that the essential cause of disease is a primary disorder of the liver; the fatty changes in the liver and kidneys have been described as resulting from a fatty degeneration of the parenchyma cells of these glands. A consideration of the pathological conditions occurring in poisoning in man and in laboratory poisoning of animals suggests an alternative explanation. It would appear to be probable that the pathological disturbance in poisoning by trinitrotoluene in particular, and also in poisoning by the five other substances mentioned, is in the first instance a disorder of fat metabolism caused by the absorption of poisons which have a special affinity for fats, with a consequent condition of abnormal lipæmia. The fatty changes in the liver and kidneys are not the results of a fatty degeneration of the cells of these glands; they represent rather a fatty infiltration, or engorgement, of the organs with fat brought to them from elsewhere. The fat in the liver represents fat brought to the liver, probably in abnormally large quantities in some cases, for purposes of normal metabolism, but which, because of functional failure of the liver, cannot be dealt with in the normal way. The fat found in the kidneys represents excessive fat in process of elimination from the blood. It appears further that the failure of the liver with respect to

its normal activity in the metabolism of fat is not caused only by overstrain of its physiological capability, but is due also to a direct action of the poison on the parenchyma cells of the gland. There is not only an excessive accumulation of fat in the liver, there is also a concentration of the poison there, brought to the liver in combination with the fat. So with the kidneys. Not only is the kidney engorged with circulating fat in process of elimination, but the cells of the gland are exposed to the action of a concentration of the poison carried there with the fat.

The Normal Process of Fat Metabolism.

Before considering in detail the nature of the disorder of fat metabolism in poisoning by trinitrotoluene it will be useful to set out briefly the general bearing of modern teaching as to the normal metabolism of fats. Fats occur in the body in three conditions: (1) as *circulating food fat* which is taken up from the intestines by the lacteals, collected in the thoracic duct, discharged from the thoracic duct into the general circulation, and subsequently altered in the liver in such a way as to be made available by various tissues and organs as a source of vital energy, (2) as *reserve fat* which, stored up in the connective tissues, represents any amount of fat ingested which is in excess of the daily need of the body, and (3) as what may be termed, in want of a better description, *available organic fat*. Available organic fat may be defined as fat, either abstracted directly from the circulating food fat or withdrawn from the reserve fat of the connective tissues, which has been rendered by the action of the liver available for the production of energy by the active tissues and organs of the body. The researches of Leathes and others have shown that the circulating food fat and the reserve fat of the connective tissues are of identical constitution, and may be recognized in the body by their low iodine value: in other words these fats contain a high percentage of saturated fatty acids. On the other hand, the available organic fats found in active organs, and in the liver in particular, have a high iodine value—their percentage content of saturated fatty acids is low. Leathes has proved further that when the reserve fat of the connective tissue is set free, or mobilized, either to meet physiological requirements or under certain conditions of disease, it is carried to the liver and is there transformed into what I have termed "available organic fat." The change effected in the liver appears to include a desaturation of the acids of the reserve fat and, probably, an elaboration of compounds of nitrogen and phosphorus with the desaturated acids. Also it is probable that so much of the circulating food fat as is required for the ordinary daily work of the body is transformed in the liver in a similar way.

Leathes has applied the results of his investigations to an explanation of the fatty changes that are found in the liver in some chronic diseases and in chloroform and phosphorus poisonings. He states the matter thus: "Too active a mobilization of stored fats, or too little activity in

dealing with them on the part of the liver, will result in an accumulation of the unfinished product in the organ. A fatty liver is the result, and the fatty acids which it contains are found to have a low iodine value." The fatty liver which occurs under the conditions is, then, a liver engorged with reserve fats, and probably circulating food fat also, which, for one reason or another, are not being transformed into available organic fat, as would happen in the normal process of metabolism. Another probable function of the liver in connexion with fat metabolism—the synthesis of fat from butyric acid derived from surplus carbohydrate food—does not appear to require consideration in connexion with the present purpose.

Microscopical Changes found in Fatal Cases of Poisoning by Trinitrotoluene.

In the course of the discussion at the Royal Society of Medicine, in March, 1917, on the causation, symptoms, pathology, etc., of "toxic jaundice" occurring amongst munition workers, the histological and other microscopical changes found in some of the organs in twenty-five cases of fatal poisoning by trinitrotoluene were referred to specially. Captain M. J. Stewart, R.A.M.C., described degenerative and cirrhotic changes in the liver in seven cases, and stated that "the amount of fatty changes varies considerably, but is never extreme." A brownish pigment, resulting —it was believed—from disintegration of the liver cells, was present generally; hæmosiderin could be demonstrated in the liver in one of the cases only. In the kidneys, "a very extreme degree of fatty degeneration is usually, but not invariably, present." Dr. Bernard Spilsbury, referring to the histological changes which he had found in poisoning by tetrachlorethane (5 cases), dinitrobenzene (1 case), and trinitrotoluene (7 cases), stated that "the effects of these three poisons are comparable in that the most marked changes are found in the liver, these consisting essentially in a fatty degeneration of the liver cells, followed by necrosis and disintegration of all the cells in large areas. These poisons also produce a varying degree of fatty degeneration in the kidneys and generally in the heart muscle."

Spilsbury found fatty changes in both liver and kidneys in all of the cases of poisoning by trinitrotoluene, and in the liver in four out of five cases of poisoning by tetrachlorethane. In the single case of poisoning by dinitrobenzene there was a coarse, irregular, cirrhotic change in the liver, and fatty degeneration was not found either in the liver or in the kidneys. Dr. H. M. Turnbull gave a very careful account of certain changes found in 11 cases of fatal poisoning by trinitrotoluene, including 7 cases (group 1) in which death was "apparently due to hepatic insufficiency," and in which severe jaundice had been associated with an extensive destruction of the liver found after death; 1 case (group 2) in which there had been multiple cutaneous hæmorrhages and slight general jaundice; and 3 cases

(group 3) in which there had been severe anæmia, without any jaundice. Turnbull stated, with reference to the first group of cases, that "the lesion in the liver may be described in general as a degeneration and necrosis of parenchyma associated with infiltration and fibrosis," that in areas of the liver in which the destruction of the parenchyma was least marked the necrosis of the cells was preceded by a fatty degeneration, and that "in areas of complete destruction of parenchyma—degenerate and necrosed, fatty, hepatic cells give place to fat granule cells." Hæmosiderin could not be demonstrated in the sections of the livers. Fatty degeneration of the cells of the kidney is described in six out of the seven cases in which this gland had been examined. The pancreas was examined in five cases, and fatty granules were found in sections of three of them. In the single case of group 2 a few "necrosed fatty cells" were found in the liver, which presented an extensive fibrotic change of the interstitial connective tissue, and there was "slight fatty degeneration of the epithelium of the second convoluted tubules and in a very few cells of the ascending limbs of Henle" in the kidneys. In two of the three cases of severe anæmia—two of which are described as cases of "aplastic, hæmolytic anæmia"—in which there was not any jaundice, a greater or less degree of "fatty degeneration" of the liver and kidney parenchyma cells is described, and there is not any description of the microscopical examination of these glands in the third case. Hæmosiderin was found in the liver in two of the cases, but does not appear to have been present in the third. Throughout the discussion at the Royal Society of Medicine, the fatty changes found in the liver and kidneys in cases of poisoning by trinitrotoluene were spoken of exclusively as representing a secondary fatty degeneration of the parenchyma cells of the glands. My own investigations suggest that, as stated above, the fatty changes in these glands result from an engorgement with fat brought to them from elsewhere.

By the courtesy of Miss Margaret Fraser, M.B., B.S., Physician to the South London Hospital for Women, I have had the opportunity of examining the liver, kidneys, and certain other organs from three fatal cases of poisoning by trinitrotoluene poisoning which were under her care in the hospital. The main clinical facts relevant to my present purpose are as follow:—

Case 1, a married woman aged 23, left work on January 18, and was admitted into hospital with jaundice on January 25. The patient became unconscious on January 26, and died on January 28.

Case 2, a married woman aged 50, left work on January 17, and was admitted into hospital on January 24 with jaundice: she became unconscious, and died on February 2.

Case 3, a single woman aged 18, left work, being then apparently in good health, on December 22. Symptoms of jaundice appeared on December 23, and the patient was admitted into hospital on December 29. The condition of the patient improved slowly, and she improved suffi-

ciently to be allowed up. At first her blood presented a normal cytological condition, the red cell count being somewhat above the normal average, and the hæmoglobin index normal. A condition of rapidly increasing anæmia became apparent later; the red cell count fell away rapidly, and the patient died with aplastic anæmia of an extremely severe type on April 18 of the following year.

It was not possible to make a complete histological examination in any one of these cases, but the following material was examined: in Case 1, the liver, kidneys and lung; in Case 2 the liver, kidneys and heart; and in Case 3, the liver, kidneys, lungs and pancreas.

The special method of fat-staining followed in the examination of sections of this material, as also in the examination of the tissues of the experimental animals, was a rapid Herxheimer method. The fresh tissues were frozen and cut. After washing in water the sections were stained, for from one to two minutes, in a mixture of equal parts of acetone and of a saturated solution of scharlach R. in seventy per cent alcohol. The sections were then washed in water, stained for one minute in the strong Mann's acid hæmatoxylin solution, washed in tap water until blued, washed in acid alcohol, washed again in tap water, and mounted in glycerine-gelatine jelly.

The changes found in the three cases respectively were as follow:—

Case 1.—The examination of sections of the liver showed advanced degeneration and disintegration of the parenchyma cells, with complete structural disorganization of the gland; a very large quantity of lightish brown granular material was scattered in small masses throughout the liver, some of the masses gave the reaction for hæmosiderin and others did not. A few collections of largish fat globules were found in some of the sections; but most of the sections were free from any trace of fat. The kidney sections showed a cloudy degeneration of the epithelium of the tubules generally, where the structure was not masked by the accumulation of fat. The straight portions of the tubules in the medullary zone contained a good deal of brownish material, varying in colour from a deep to a light greenish brown. The convoluted and straight portions of the tubules showed thickly numerous granules of fat, both in and outside the epithelial cells. To the naked eye the lungs appeared to be in a condition of solid œdema. Microscopically, there was marked cellular infiltration of the connective tissue stroma of the lungs, without any marked exudation into the alveolar spaces. Scattered through the interalveolar tissues there were numerous fat granules, some lying apparently free in the tissues, others contained in largish mononuclear cells. Small masses of brownish material occurred here and there in the interalveolar tissue.

Case 2.—Sections through the liver showed advanced interlobular round-celled infiltration and fibrosis, with marked degeneration of the parenchyma cells generally. There was a large amount of fat, distributed in globules of various size, partly in the interlobular round-celled and

fibrotic tissue, and partly lying either in or between the parenchyma cells, especially about the periphery of the lobules. Numerous small masses of brownish pigmented material were scattered throughout the liver, only a relatively small proportion of which stained as haemosiderin. A large quantity of fat was present in the kidney. As in Case 1, the fat was confined to the convoluted and straight portions of the tubules in the cortical zone, where globules of fat could be seen lying between the tubules, in the epithelial cells, and a little in the lumen of the tubules. As in Case 1 also, the glomeruli were free from any traces of fat. Here and there a tubule contained dark brownish or greenish brown material. The heart muscle showed the appearances commonly associated with "pigmentary degeneration," or brown atrophy; but there was not any abnormal fat in the sections stained with scharlach R.

Case 3.—The parenchyma cells of the liver were mostly in a stage of advanced degeneration. There was a good deal of accumulated fat, especially about the central zone of the lobules; the exact relation of the fat to the remains of the parenchyma cells could not be determined. A large quantity of lightish brown material, none of which gave a hæmosiderin reaction, was scattered throughout the sections. The kidney section showed an advanced degenerative change of a hyaline appearance throughout the tubules. No fat in connexion with the tubules themselves could be demonstrated with the stain, but a very few minute fat globules could be seen lying either in or between the cells of the glomeruli. Some of the tubules contained casts of a reddish-brown homogeneous material. The sections of the lungs were of normal appearance, and did not show any fat. The pancreas showed an extensive fibrosis of the interstitial tissue, with advanced degeneration of the parenchyma cells, which could not be differentiated individually. In sections stained with scharlach R. the widely isolated lobules appeared as islands of pigmented, and more or less disintegrated, material lying in a fibrotic mass, and full of globules of fat of various sizes.

The Interpretation of the Significance of the Fatty Changes found in "Toxic Jaundice."

Experiments carried out some years ago in connexion with the action of poisonous doses of ether and chloroform on the rabbit suggested that the fatty changes appearing in the liver could not represent a primary fatty degeneration of the parenchyma cells of the gland. Under the experimental conditions in the lower animals, and equally in cases of delayed chloroform poisoning in man, the accumulation of fat is greater in amount, and more rapid, than could be accounted for in this way. And Leathes' hypothesis, applied to cases of phosphorus and chloroform poisoning in man, appears to supply satisfactory alternative explanations. The fatty changes in these cases do not represent any degenerative change in the parenchyma cells themselves, but result from infiltration of the liver with

fat carried to the gland from elsewhere. And the accumulation is either because there is an abnormal liberation of the stored connective tissue fat, or because the functional activity of the liver is so impaired that the gland cannot deal with the ordinary food-fat. Or, as appears to be the case in poisoning by trinitrotoluene and some other substances, the two factors are working together—there is an abnormal mobilization of the stored fat, and the functional activity of the liver is impaired by the direct action of the poison on the parenchyma cells with consequent degeneration, but not of a fatty kind. Considering the condition found in man in poisoning by trinitrotoluene, it does not appear that there is any correlation between the degree of fatty change in the liver and kidneys and the degree of actual degeneration of the cells of these glands. In Case 1 there was advanced degeneration of the liver parenchyma cells with very little fatty change. The kidney was engorged with fat, but there was not any marked alteration of the epithelium in the medullary portion of the kidney, where the structure of the cells could be seen clearly without the complication of the picture caused by fat engorgement. In this case it may be supposed that the liver had become functionless, in the matter of fat metabolism, whilst the fat taken with food was being eliminated, probably unchanged, by kidneys which were still moderately effective functionally. In Case 2, an abnormal lipæmia was indicated by the presence of fat in the interstitial tissue of the lungs, the parenchyma cells of the liver were less completely destroyed, and a certain amount of fat was still collecting in the liver, and apparently was being passed—imperfectly—through the cells of the lobules from the periphery towards the central vein. Meanwhile the excess of food-fat was being eliminated, as in Case 1, by the kidneys. The circumstances of Case 3 appear to be altogether different. In this case the patient had recovered, it may be assumed, from the direct action of the trinitrotoluene; she died of sequelæ to the acute poisoning, the principal cause of death being an aplastic anæmia, due to destruction of the bone-marrow at an earlier stage of her illness. The liver cells had undergone extensive degeneration, but the gland still held a moderate amount of fat. The absence of fat from the kidneys was remarkable in this case: possibly the amount of absorbed food-fat had been reduced to very low proportion in consequence of destruction of the activity of the pancreas which, judging by the extent of the fibrotic change and the degeneration of the parenchyma cells, must have been nearly or quite functionless. It was noticeable, also, that together with an absolute absence of fat from the tubules of the kidney there was a far-advanced degeneration of the renal epithelium. The biological mechanism of the degeneration of the pancreas, which has been observed in other cases of poisoning by trinitrotoluene, cannot be explained at present.

Experimental Poisoning with Trinitrotoluene: Cats and Rabbits.

The findings in the three cases referred to, together with a consideration of the pathological changes described by others, would appear to justify the assumption that there is a degeneration of the parenchyma cells of the liver, and in certain cases of the kidneys also, caused by the direct action of the poison. The degeneration thus caused is not a "fatty degeneration." When the degeneration of the liver cells has reached a certain point, a natural sequence will be, in accordance with what has been pointed out with regard to the normal fat metabolism, the establishment of an abnormal lipæmia caused by an excess of circulating food fat. Beyond all this there is, however, the question as to whether there is not in poisoning by trinitrotoluene and the other poisons under consideration a further disturbance of fat metabolism, resulting from a freeing, or mobilization, of the reserve fat of the connective tissues, whereby the condition of lipæmia is intensified.

In an attempt to clear up this point, the action of trinitrotoluene, administered by the mouth by subcutaneous injection and by inunction into the skin, has been tested on the cat and the rabbit. Because of circumstances the number of experiments has been limited, but the results obtained appear to be closely relevant to the question.

Cat I., weighing 4,750 grammes, was poisoned rapidly with trinitrotoluene administered by the mouth, 0·75 gramme being taken with food at 3 p.m., and 0·5 gramme exactly twenty-four hours afterwards. The animal did not show any signs of poisoning after the first dose; but at 10 a.m. on the third day, nineteen hours after the second dose, it was found crouched up in its cage in a semi-comatose condition. It became deeply comatose about an hour later, lying extended on its side. There were occasional convulsions, and the animal died at 4 p.m., forty-nine hours after the administration of the first dose. Examination after death: The body weighed 4,350 grammes, and all the organs were of natural appearance. The animal had not passed any urine during the last day; the bladder contained about 0·5 cubic centimetre of turbid, almost black, highly concentrated urine, which had a very disagreeable odour. With Webster's test the urine gave a characteristic reaction for trinitrotoluene in organic combination. Sections of the liver, kidneys, and spleen were stained for microscopic examination. The liver showed a slight, but quite definite, degree of interlobular round-celled infiltration. The parenchyma cells appeared to be of a brownish colour, and did not take the hæmatoxylin stain well. A few globules of fat, stained by scharlach R., were scattered through the sections, some lying within the lobules, and some in the areas of interlobular round-celled infiltration; some of the fat was contained within cells, some was apparently free. There were not any pigment masses in the gland. The cortical portion of the kidneys was

heavily engorged with fat, so that sections stained with scharlach R. presented a bright red cortical zone when looked at with the naked eye. On microscopic examination it was found that the Malpighian capsules were free from fat, but the rest of the cortical portion of the kidney was so crowded with largish globules of fat that the relations of the fat and the tubular epithelium could not be defined. The straight tubes in the medullary portion of the gland contained some brownish material which did not stain as hæmosiderin. In sections which included the area of the pelvis of the kidney, some globules of free fat could be seen; this fat, however, might have been displaced from the cortical zone in making the sections. The spleen was of normal appearance, the pulp stained well with hæmatoxylin, and neither fat nor abnormal pigment were there. The symptoms in the case and the apparent blockage of the kidney tubules with fat suggested uræmia as the immediate cause of death.

Cat II., weighing 3,750 grammes, was given occasional doses of trinitrotoluene, 0·5 gramme every third or fourth day, mixed with lean meat or fish, for a period of about three weeks. Very little of the poison, however, was actually taken; after swallowing a mouthful or two of the food, an attack of sneezing would come on, followed by salivation, and unmixed food had to be given. The exact amount of trinitrotoluene ingested could not be estimated, but at the end of the three weeks the animal appeared to be in perfect health, and had not lost any weight. Attempts to administer the trinitrotoluene by the mouth were stopped, and the cat was put on a diet of lean meat. The skin of the right flank was then shaved over an area of about fifteen square inches and one gramme of trinitrotoluene, mixed with vaseline, was rubbed thoroughly into the shaven skin on each of three consecutive days. Three more doses were rubbed into the skin with two-day intervals, so that six doses of one gramme each were applied during a period of twelve days. On the thirteenth day the animal was very drowsy, would not take its food, and appeared to be moribund. Its condition improved on the following day, and on the fifteenth day it had apparently recovered, and was taking its food well. Another dose of one gramme was applied during the afternoon, and the animal was found to be in a drowsy condition two hours afterwards. On the following day the drowsiness had passed off—the animal was taking its food, and another dose of one gramme was rubbed in. On the next day, the seventeenth day after the commencement of inunction, there was extreme drowsiness, and the hind limbs were paralysed; a dose of 1·5 gramme was applied. The animal died on the eighteenth night after the commencement of the inunctions, and after 9·5 grammes had been thus administered. Examination after death: The body was much emaciated, weighing only 1,150 grammes. The organs were of natural appearance, except that the liver appeared to be rather deeply congested. The bladder was distended with urine, which was of acid reaction and of a dark brown colour. At first the urine appeared to be quite clear, but, on standing, a mucus-like

sediment began to separate out. On centrifugalizing the urine, a somewhat dense sediment of mucus was obtained, in which were entangled a very large number of globules of fat, which measured from about 0·5 micron to twenty microns, and which stained in a typical way with scharlach R. A strong reaction was obtained with Webster's test for trinitrotoluene in organic combination. The nature of the dark brown pigment in the urine was not determined; so far as could be decided by chemical and spectroscopic examination, it was not a hæmoglobin derivative. The microscopic examination of the skin will be referred to later on. Sections of the liver showed a well-marked round-celled infiltration of the stroma, with degeneration of the parenchyma cells, many of which, however, still stained well with hæmatoxylin. There was a large quantity of dark brownish material infiltrating the lobules, and collected especially round the central vein, which in some instances appeared to be plugged with the material. The cortical zone of the kidneys contained an enormous quantity of fat in small granules or globules, which appeared to be situated both in and between the epithelial cells of the tubules. Granules of brownish material were present either in, or on, the epithelium of the Malpighian capsules, which were free from fat. Brown pigmented material could be seen in the straight tubules of the medullary portion. A quantity of brown pigment was distributed throughout the lungs, between the air vesicles; in some places small vessels, seen in transverse section, were apparently plugged with this material. Sections through the heart muscle showed a brown pigment distributed between the muscle fibres and in places apparently in the capillaries. The spleen pulp was heavily loaded with brown pigmented material, which, however, was present very scantily in the Malpighian bodies. None of the pigment in the liver, kidney, or spleen stained as hæmosiderin.

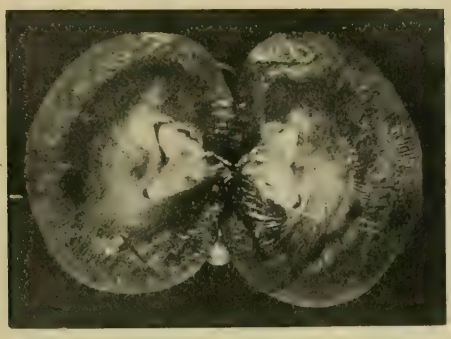
Cat III, weighing about 3,500 grammes, was fed for seventeen days with occasional doses of 0·5 gramme of trinitrotoluene mixed with various foods—lean meat, fish, cream and margarine. But difficulties similar to those which prevented the carrying out of feeding experiments with Cat II occurred. After a small quantity of food had been taken, fits of sneezing, followed by salivation, came on, and at each meal unmixed food had to be substituted after quite a small quantity of trinitrotoluene had been ingested. The cat remained apparently in good health, and had not lost weight when it was killed, eighteen days after the commencement of the experiment. Examination after death: All the organs were of natural appearance. The bladder was empty of urine. The liver showed commencing round-celled infiltration of the stroma, the parenchyma cells stained rather badly with hæmatoxylin, and there were a certain number of fine globules of fat scattered sparsely through the sections. Sections through the kidneys showed a moderate amount of fat in the cortical zone. The epithelial cells of the tubules in the cortical zone were of normal appearance and in places were stained a diffuse pinkish colour by scharlach R., the bright red-

stained globules of fat, mostly in the part of the cell remote from the lumen of the tubule, standing out in sharp contrast. There was a considerable quantity of brown homogeneous material in the tubules, especially in the convoluted portions, and a little amongst the epithelium of the capsules of the glomeruli. The vessels of the kidney were overdistended with blood, and blood cells in addition to the homogeneous brown material were present in the tubules and in the Malpighian capsules in places. Sections of the spleen showed a considerable quantity of brown pigment scattered through the pulp, the Malpighian bodies here and there showing largish masses of it.

Rabbit I, weighing 2,260 grammes, was fed by means of an œsophageal tube with considerable quantities of trinitrotoluene; altogether 8·4 grammes were given, in twenty-one doses varying from 0·25 to 0·6 gramme, during a period of thirty days. The animal remained in good health and took its food well throughout the period; it lost weight at first, but at the end of the experiment weighed the same as at the commencement. On the day after the feeding with trinitrotoluene was stopped, a small area of the skin was shaved, and 2 grammes of trinitrotoluene, mixed with vaseline, were rubbed in. On the next day the animal was very drowsy, and did not take its food, but had quite recovered on the day following. On each of the next two days a dose of 1·75 gramme was rubbed into the skin. The animal became drowsy again after the last inunction, and was killed on the following day. Examination after death: All of the organs appeared to be healthy. The bladder contained a small quantity of acid urine of a dark coffee-brown colour. A considerable sediment, consisting mainly of long renal casts of homogeneous dark-brown material, was thrown down on centrifugalization of the urine. Sections through the liver showed a quantity of fat, especially in the zone round the central vein of the lobules. The parenchyma cells of the gland were apparently but little affected. There were indications of commencing round-celled infiltration of the interstitial tissue. The epithelial cells of the kidney tubules appeared to be swollen and in many places in the cortical zone appeared of a brown tinge. Many of the tubules were filled with homogeneous dark brown material; small masses of similar material were present on the capsule of the glomeruli here and there. No fatty change was apparent. Blood cells were seen in some of the tubules. The spleen was loaded with fine and coarse granules of brown pigment. Sections of the lungs showed a number of cells containing minute fat granules in the interalveolar tissue which appeared to be increased in bulk, or œdematous. The pigmented material in the spleen and kidneys did not stain as hæmosiderin.

Rabbit II, weighing 2,050 grammes, was injected with a suspension of trinitrotoluene in normal saline solution, thirteen doses varying from 0·1 to 0·45 gramme being given subcutaneously during a period of twenty-two days. Because of the rapid separating out of the suspension in normal saline solution the exact dosage was difficult to determine, but it was

estimated that about 4·75 grammes, possibly a little less, were administered by the thirteen injections. The animal remained apparently quite well throughout the experiment, except that sometimes it became drowsy after an injection. It took its food well and put on weight at first, to the amount of 200 grammes by the end of the first eight days. After then the weight gained was lost gradually, but just before the animal was killed, on the day after the last injection, the weight, 2,100 grammes, was a little more than the weight at the commencement of the experiment. Examination after death: The body was kept in cold storage for a few hours before it was examined. All the organs were of natural appearance externally. On cutting the kidneys open, it was found that the pelvis of each gland was filled with a solid plug of fat (*see* illustration). The bladder contained a large quantity of clear acid urine of a dark coffee-brown colour. On extracting the urine with ether, the ethereal extract was of a light reddish brown colour, and did not give the reaction for trinitrotoluene with



Webster's test. The urine residue was then acidified with sulphuric acid, and extracted with ether. The second ether extract was of a light yellow colour, and gave an intense reaction with Webster's test. Sections of the liver showed a small amount of interlobular round-celled infiltration, with small globules of fat in the interstitial tissue here and there. The parenchyma cells stained well with hæmatoxylin, and did not show any marked changes. There was not any fat within the lobules, it being present only amongst the cells of the infiltration occurring along the course of the interlobular portal circulation. A few small collections of pigmented matter were seen in some of the sections. The cells of the cortical zone portions of the kidney tubules were stained a pinkish colour by scharlach R., and a certain quantity of fat globules, stained typically, were present. Sections through the gland showed a solid plug of fat free in the pelvis of the kidney. The spleen was loaded heavily with dark brown pigment, which did not stain as hæmosiderin. The sections through the lungs showed normal appearances, excepting that there were small collections of brownish pigment here and there.

For control purposes, in connexion with the animal experiments, a series of sections through the lung, liver, kidney, and spleen of a healthy cat and of two healthy rabbits were cut and stained with scharlach R. under conditions exactly similar to those under which the sections from the organs of the experimental animals were prepared for examination. In the liver of the normal cat a few isolated large globules of fat, and a few small collections of small globules, were scattered at considerable distances apart through the sections. In sections through the kidneys, a good deal of the epithelium of the portions of the tubules within the cortical zone was stained a diffused light pinkish colour, and small droplets of red-stained fat could be seen in the epithelial cells, but in small numbers and widely separated. It is well known that fat occurs, sometimes in noticeable quantities, in the kidney of the healthy cat; but there could not be any comparison between the presence of the comparatively small amount of fat in the gland of the normal animal and the heavy flooding of the kidneys of the experimental animals with fat. There was not any fat in the lungs of the healthy cat, except a little in the connective tissue immediately around some of the larger bronchioles; nor was any deposit of pigment found in any of the organs examined. Neither fat nor pigment could be found by means of the method employed in any of the organs from the two normal rabbits that were examined.

Effect of Poisoning by Trinitrotoluene and Certain other Substances as regards Fat Metabolism.

With respect to their special affinities for fats the six poisons under consideration may be divided, somewhat artificially, into two classes. Three—tetrachlorethane, chloroform and ether—are active solvents of fats; the other three—trinitrotoluene, dinitrobenzene, and phosphorus—are readily soluble in fats. An excess in the blood of one of the first class of poisons, following inhalation of the vapour, will probably act directly on the stored fat of the connective tissues, with the production of an excessive lipæmia—excessive in the sense that the mobilization of reserve fat is not in response to a physiological requirement. The liberated fat will accumulate in the liver, its natural destination for the purpose of being converted into "available organic fat" carrying with it the substance which has liberated it. More of the poison will be brought to the liver with the circulating food fat. The assumption that the fixed fat of the connective tissues, as well as the circulating food fat, may be affected by the presence of, for instance, chloroform which has been taken up by the blood, is founded on the relatively rapid accumulation of fat in the liver and kidneys in the experimental poisoning of rabbits with chloroform. The engorgement with fat is sometimes so intense that it is difficult to believe that all the fat present could be provided, within a few days, by the ordinary diet of laboratory animals. When trinitrotoluene which has

been fixed in the skin is taken up, in one way or another, by the blood it appears to be probable that there is a similar process, connective tissue fat and circulating food fat being affected alike. When poisons of the second class—trinitrotoluene and phosphorus more especially—are swallowed, their special affinity for fats will cause concentration of them also in the liver. The poison will be dissolved in the fatty constituents of the intestinal contents, will be taken up with the fats by the lacteals, and will be carried directly to the liver with the circulating food fats. The picture of poisoning in all these cases represents, therefore, combination of the poison with fat, abnormal liberation of fixed connective tissue fat, concentration in the liver of the poison in combination with the fat, impairment of the fat-metabolizing and other functions of the liver, an excessive accumulation of fat in the blood, and engorgement of the kidney with that fat in process of elimination from the blood. And so the excretory cells of the tubules of the kidney, as the parenchyma cells of the liver, are subject to the effect of a concentration of the poison carried to them with the excess fat in the blood. One effect of the fixation of trinitrotoluene in the fat of the connective tissue is illustrated conspicuously by the aplastic anæmia which occurs occasionally amongst the special class of munition workers affected, as an effect of the action of the concentrated poison on the hæmatogenic cells of the bone marrow.[1] Evidence of an excessive lipæmia was furnished by the presence of fat in the interalveolar connective tissue of the lungs in one of the cases of human poisoning, and in one of the two experimental rabbits. Similar evidence was furnished by the finding of fat in the areas of round-celled infiltration of the liver, in addition to the fat occurring within the lobules. The fact of elimination of fat by the kidneys, under the conditions of experimental poisoning, was proved by the finding of large quantities of fat in the urine of one of the experimental cats, and by the solid plugs of fat found in the pelvis of each kidney in the case of one of the experimental rabbits. In these cases, then, there are two main pathological processes to be taken into account, the effect of the poison on fat-metabolism, and the direct action of the poison on various organs—the liver and kidneys more particularly. And, it may be repeated, the degenerative changes produced in the parenchyma cells of these organs by the direct action of the poison are not of the nature of a fatty degeneration.

In certain other experiments carried out in the Department in which

[1] The fact that the obvious destruction of red cells which occurs in poisoning by trinitrotoluene does not cause any marked secondary anæmia, in the majority of cases, is of interest in connexion with the special affinity of the poison for fat. It is suggested that the fixation of a certain moderate amount of trinitrotoluene in the bone marrow causes a stimulation effect, with an increased output of red cells which compensates for their destruction. In comparatively rare cases the action of the trinitrotoluene goes beyond the point of stimulation, destruction of the bone marrow as a cell-producing centre results, and aplastic anæmia follows.

rabbits have been poisoned with arseno-benzene compounds, which closely resemble trinitrotoluene in some of their pathological effects, it has been found that the first appearance of fat in the liver is not in the parenchyma cells, but in the areas of interlobular round-celled infiltration which appear along the course of the portal circulation. In such cases there may also be evidence of elimination of fat by the kidneys.

The Origin of the Pigments found in the Urine and in Internal Organs in Poisoning by Trinitrotoluene.

The question of the origin of the pigments occurring in the urine and distributed through the liver, kidneys, spleen, lungs, and other organs in poisoning by trinitrotoluene—whether in human cases or in the experimental poisoning of lower animals—is complicated by the fact that two different kinds of organic pigment at least, and probably three, have to be considered and differentiated. In the first place there is, undoubtedly, a residual blood pigment produced by the action of the poison on the hæmoglobin of the blood cells, the first stage in the production of the pigment being marked by the presence of methæmoglobin in the blood, and a later stage by the presence of hæmosiderin in the liver and spleen. Secondly, there are the bile-pigments, manifested by the jaundice of human cases. And for the third kind of pigment, it appears from observations made in the course of my investigation that a brownish benzene derivative pigment is produced in the body in poisoning by trinitrotoluene. It appears to be probable also that a similar pigment is produced in certain cases of poisoning by arseno-benzene compounds—cases which present otherwise a remarkable resemblance in essential respects to cases of poisoning by the benzene derivative, trinitrotoluene. It can only be suggested now that this organic pigment may be related to that produced in the urine in cases of phenol poisoning by the oxidation of hydroquinone or p-dioxybenzene.

The direct action of trinitrotoluene on the red blood cells was studied by microscopic methods in connexion with sections made across the area of the skin of Cat II into which the poison had been rubbed. These sections showed a dilatation and thrombosis of many parts of the superficial cutaneous capillary system with plugs of red blood cells. In these places the blood cells were of a distinctly brownish colour. Also it appeared as though a process of disintegration of the cells of the minute thrombi was in progress in places. If in absorption of trinitrotoluene by the skin any quantity of the poison is fixed in the red cells of the cutaneous capillary circulation, it is at least possible that the effects of the concentration of fat-carried poison on the liver are accentuated by a certain further concentration of the poison in the gland, carried by red cells which are undergoing their ultimate stage of disintegration there.

Channels of Absorption in Poisoning by Trinitrotoluene.

The method of absorption of fat-carried trinitrotoluene from the intestine has already been referred to. The degree of danger connected with the direct inhalation of dust into the lungs is not known, but is probably not considerable. Nor does it appear probable that there is any appreciable danger arising from inhalation of trinitrotoluene in vapour at ordinary temperatures. The danger arising from absorption of trinitrotoluene through the skin is well recognized, and would appear to be proved directly by the observations made in respect of the skin of Cat II. The fixation of the poison by fatty, or fat-derived, constituents of the skin is of special interest in relation to "late" poisoning by trinitrotoluene, in which symptoms of illness may first manifest themselves several weeks after the last exposure to the poison. This late cumulative effect occurs, so far as I am aware, in only one other recognized class of poisoning—that caused by arseno-benzene compounds. In the case of poisoning by trinitrotoluene, the late action has been explained by assuming a cumulative effect of small quantities of the poison absorbed gradually from the skin. The explanation is not altogether satisfactory. It is not clear, by any means, that a poison fixed in the superficial fatty constituents of the skin, themselves in the way of elimination from the surface of the skin, would be taken up and carried internally. On the other hand, we know that blood thrombosed in the cutaneous capillaries, as happened in the experiment on Cat II, will be absorbed gradually, and with it the benzene poison. It may be, therefore, that the red blood cells have an effective part in these cases. It has been said that it does not appear to be probable that any of the poison which has become fixed in the fatty constituents of the skin will become absorbed directly from the skin, but rather will ultimately be eliminated from the surface. At the same time it is not certain that trinitrotoluene fixed in the skin in organic combination may not be absorbed in another way, indirectly by inhalation. It is known from recorded experiments that trinitrotoluene, as such, is volatilized freely only at relatively high temperatures, and that animals exposed to such volatilized trinitrotoluene are not readily affected. But it is doubtful as to how closely these experiments bear on the conditions prevailing when the trinitrotoluene has become fixed in the skin in organic combination, and possibly with some alteration of its constitution. It may be that the fixed poison is volatilized slowly from the skin at the body temperature, so that the patient—when warm in bed, for instance—is continually inhaling small quantities of the vapour.

The Prevention and Treatment of Poisoning by Trinitrotoluene amongst Munition Workers.

It remains to be considered to what extent the views expressed as to the pathology of poisoning by trinitrotoluene are applicable to measures for prevention and treatment. Little need be said as to the general

administrative measures carried out to protect the worker from undue exposure to the action of the poison. Such measures have been planned skilfully, and carried out with, it is understood, a large measure of success. One point, however, in connexion with the protection of the individual worker must be referred to particularly. Because of its high fat-content, and in view of the ready solubility of trinitrotoluene in fats, milk—as all other foods of a fatty kind—should be avoided as much as possible. Fatty foods, in effect, will favour the absorption of the poison from the intestine. The fatty constituents of the munition worker's dietary should be replaced, as far as may be practicable, by sugars—by glucose and by fructose supplied in fruit foods. The elimination of any trinitrotoluene already fixed in the fatty constituents of the skin should be promoted by frequent warm baths, and especially vapour baths, followed by brisk friction of the skin. Equally, the administration of milk, or any other fat food, in the course of the treatment of declared poisoning is inadvisable. Whatever may be the exact causation of the disease of the liver, two facts are outstanding. In the first place, the liver functions become seriously disordered, so that the gland is no longer able to carry out effectively its part in fat metabolism. In the second place, a condition of excessive lipæmia is established. Under these conditions all fats should be cut off from the dietary, so that the effects of the existing lipæmia may not be aggravated, and so that the liver may be relieved of its metabolic work as far as may be. As in preventive treatment sugars should be substituted for fat foods, definite doses of glucose being given together with fruits. The sugar given thus is given not only in substitution for fatty foods, but also because of theoretical considerations which suggest that oxidation of the excessive floating fat will be favoured by its administration. The diet then should consist mainly of carbohydrates, with very small quantities of lean meat, or preferably fish—excluding the fatty kinds, such as herring, mackerel, etc. It has been suggested that the natural elimination of trinitrotoluene fixed in the skin is outwards. Therefore, apart from the removal of the fixed poison by means of chemical solvents, the free use of warm vapour baths, followed by friction, is indicated from the very commencement of illness. Vapour baths have been used extensively in the treatment of poisoning, but mainly for another purpose and in the later stages when symptoms of coma are threatening.

In conclusion, I have to express my thanks to Miss Margaret Fraser, M.B., B.S., Physician to the South London Hospital for Women, who has kindly allowed me the use of material from cases under her care, and to Miss Hilda K. Whittingham, M.B., B.S., D.P.H., Bacteriologist in the Hygiene Department, Royal Army Medical College, who has assisted throughout the work.

THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES AS OBSERVED IN WARFARE.

By Lieutenant-Colonel GORDON HOLMES.

Royal Army Medical Corps.

(*Continued from p.* 475.)

(*C*) *The Rebound Phenomenon.*—Several years ago in a paper written in collaboration, Dr. Grainger Stewart and I pointed out a sign of cerebellar involvement that is of considerable importance in the interpretation of the disturbances of voluntary movement produced by this condition. The patient's elbows are supported on a bed or table and he is asked to pull each hand in succession towards his mouth against resistance offered by the observer who grasps his wrists; when this resistance is suddenly released the hand of the affected side flies to his mouth or shoulder, often with considerable violence, but the movement of the normal limb is arrested almost immediately by a contraction of the antagonists (triceps), and may even be jerked back or rebound. The rebound is excessive in spastic limbs.

The absence of this rebound may be demonstrated in various other actions, as extension of the elbow, elevation or depression of the arm at the shoulder, flexion or extension of the hip and extension of the knee. It occurs only on the same side as the lesion when this is unilateral, but it is seen on both sides when the wound is bilateral. It is only necessary to resist any movement which the patient attempts to perform and remove the resistance suddenly: then the limb or the segment of the limb which he wishes to move swings unchecked or excessively in the direction of the movement attempted and is not quickly arrested as the normal invariably is. If a hand is placed on the antagonistic muscles it is found that these do not contract, or they come into contraction too late.

The occurrence of this "rebound phenomenon" in lesions of the cerebellum has been confirmed by other observers (Bing, André-Thomas) and it was easily demonstrable in all cases of my present series in which there were moderate or severe lesions. As a rule it was equally pronounced at all joints, though it was usually more definite in the upper than in the lower limb, and more easily evoked at the proximal than at the distal joints. It often happens that it becomes less easy to demonstrate after it has been tested for several times, owing to a voluntary effort on the part of the patient to suppress it, especially if the unchecked movement may produce pain or discomfort.

A similar inability to arrest passive movement quickly may be seen if the observer supports the elbow with one of his hands, while with his

other he first moves the patient's forearm about aimlessly and then unexpectedly throws the hand towards his face. The passive flexion of the normal arm is quickly checked, but the affected hand flies, often violently, into the patient's face, just as a flaccid paralysed limb or a loosely-hinged rod would.

Further, if when his elbows are supported, the patient attempts to flex and extend his forearms alternately as quickly as possible the range of movement on the affected side is often excessive, and more than one patient has complained that he has actually jarred or hurt his elbow-joint by the excessive flexion, which is arrested only by the structure of the joint or by its ligaments, and not by contraction of the antagonistic muscles.

This phenomenon is always most pronounced in the early and acute stages of a cerebellar injury and diminishes gradually as improvement sets in. It is consequently more prominent when the limbs are hypotonic, but its relation to hypotonia will be discussed later.

(*D*) *Adiadochokinesis.*—Babinski originally pointed out that a patient with cerebellar disease is almost always unable to execute alternate movements as quickly and correctly as the normal person, and he termed this symptom adiadochokinesis. It may be tested at any joint at which such movements can be easily and rapidly performed, as flexion and extension, or pronation and supination of the elbow, or flexion and extension of the fingers, ankles, or toes.

When a patient with a unilateral cerebellar injury is asked to pronate and supinate his forearms alternately and as rapidly as he can a very striking difference is noticed between the movements on the two sides, as the rate of the alternate movements of the homolateral limb is slower and much less regular, their range is less uniform, and both the slowness and the irregularity in rate and range become more pronounced the longer the effort is continued. In slight injuries the awkwardness of the attempt and the apparently greater effort necessary for it on the patient's part are more striking than the slowness of its execution, and in very limited lesions or when recovery sets in only this awkwardness and incompleteness may be observable.

Further, while the other joints of the normal limb remain fixed, on the affected side various adventitious movements occur, as irregular flexion or extension of the fingers and more particularly flexion and extension of the elbow, or adduction and abduction at the shoulder. These are generally obvious at once, but if not pronounced the observer by placing his hands under the elbows can feel that while that of the unaffected arm remains steady, the other is jerked about irregularly. Similar adventitious movements at the wrist and elbow can be often observed when rapid flexion and extension of the fingers are attempted, and in the knee and ankle when he is asked to move his toes only.

If the limb is hypotonic part of the adventitious movements may be attributed to flail-like flopping of its distal segments; when for instance the elbow is rapidly flexed and extended the wrist swings about inertly, flexing as the hand is brought towards the shoulder and extending as the elbow is rapidly straightened; and in the same test the elbow is often raised from its support by the momentum of the flexing arm. They depend partly therefore on the failure of the fixing muscles to preserve the correct posture of those segments of the limb that should not move. But there can be no doubt that they are largely due to the active contractions of muscles that are not directly concerned in the act attempted by the patient: this can be easily determined if the arm and shoulder muscles are carefully palpated while rapid pronation and supination of the forearm is attempted; or if a hand is placed on the thigh as the patient attempts to flex and extend his toes rapidly the irregular contractions of its muscles, which produce movement of the knee, can be felt. There is consequently a tendency to an irregular spread of the innervation to other muscles than those which under normal conditions execute the act. When the test is prolonged the movements which the patient wishes to perform may be in fact largely replaced by irregular aimless displacements of other segments of the limb.

Occasionally even the active muscles do not work together accurately and harmoniously, and this increases the awkwardness and irregularity of the patient's attempts; in rapidly flexing and extending the fingers for instance, all these at first move together as they invariably do in the normal limb, but after a time they frequently get out of line and cease to flex and extend simultaneously or to the same extent. I have even seen the thumb bent involuntarily into the hand and thus interfere with the excursions of the fingers.

The slowness and irregularity can be equally well seen in more complex actions, as in shaking hands, clapping hands, rubbing a mark off a sheet of paper, stamping his feet, etc. When a patient attempts to clap hands both arms generally move at first, but the affected one soon comes to a standstill and is then only passively displaced by the blows of the normal hand.

The causes of this disturbance can be more easily analysed if some simple action, as tapping a table or the observer's hand, is carefully examined. In the first place the range of movement is irregular; his hand is occasionally raised too high or brought down too firmly, but more commonly it is smaller than that of his normal limb. Often indeed the movement is arrested too soon and the hand may not actually come to the table. The slowness is due chiefly to delay at the turn and not to time lost in the movements themselves, for there is rarely much difference in the rate of these and of those of the normal limb.

The principal features of adiadochokinesis may be seen in figs. 8 and 9. Fig. 8 reproduces tracings of alternate pronation and supination of the forearms, in a patient with a right-sided lesion in whom this symptom

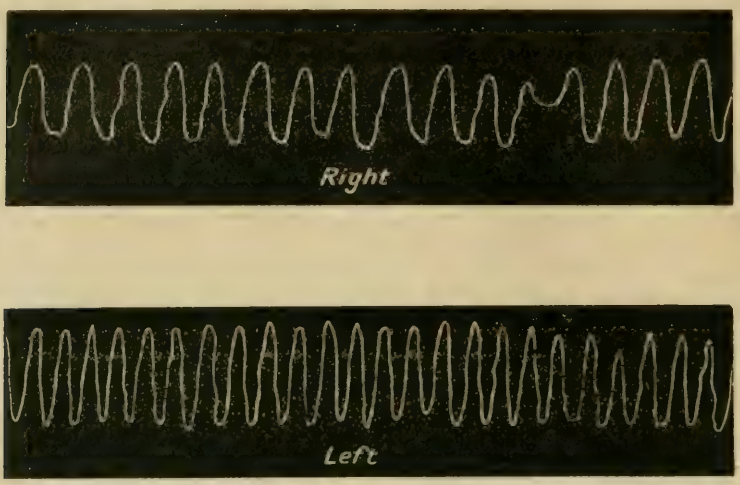


FIG. 8.—Adiadochokinesis. Tracings of pronation and supination of the forearms in succession when performed as rapidly as possible by the patient, in a man five weeks after the onset of a moderately severe right-sided lesion.

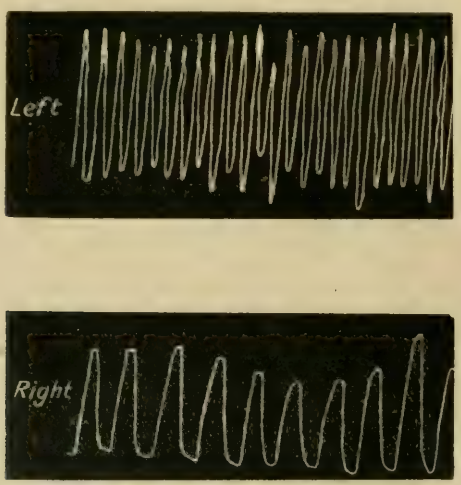


FIG. 9.—Adiadochokinesis. Tracings of alternate flexion and extension of the fingers performed in succession as rapidly as possible by the patient; flexion is represented by the downward lines, extension by the upward. From a man forty-one days after the infliction of a severe right-sided cerebellar wound.

was rapidly disappearing. The range of the excursions of the affected (right) arm was fairly regular, though on one occasion the movement was checked in semi-pronation, but it was considerably smaller than that

of the normal limb; the slower rate was due chiefly to time lost at the turn and to slowness in the initiation of each sequence of muscular contractions. In fig. 9 tracings obtained from rapid flexion and extension of the fingers are reproduced; here the same features are present, but the delay at the turn of the movement, and especially in extension of the fingers, is more obvious.

The occurrence of adventitious movements certainly contributes to the awkwardness and slowness, since they confuse the patient in his efforts, and often put the contracting muscles at a mechanical disadvantage. Consequently we can conclude that the condition described by Babinski as adiadochokinesis is due to disturbance of the range of movement and to a slowness in initiating each excursion of the limb, while the occurrence of adventitious movements is partly due to lack of proper co-operation on the part of those muscles which should fix other joints of the limb, and partly to irregular purposeless contractions of other groups of muscles.

Frequently such alternate movements can be better performed by the affected limb when it is tested alone than when both limbs are examined simultaneously. The normal person generally finds it easier, or at least as easy, to perform such rapid alternate movements simultaneously with his right and left limbs as with either separately, but when a unilateral cerebellar lesion exists the more rapid movements of the normal limb seem to confuse those of the affected one and disturb their sequence. The patient has, consequently, to attempt two different actions at once instead of the same action simultaneously with his right and left limbs.

Adiadochokinesis can usually be demonstrated at all joints of the affected limbs, even when only limited lesions exist, but it is usually more pronounced in the arm than in the leg and in complex than in simple actions. During recovery the slowness, irregularity and awkwardness diminish gradually, but adiadochokinesis is one of the most persistent of the ordinary signs of cerebellar injury.

The majority of the abnormalities of movement described above refer to those in which the larger and more proximal groups of muscles are concerned, but similar disturbances can be observed in the finer and more elaborate actions of the hand which are peculiar to the anthropoid and especially to man. The slowness, awkwardness and irregularity of the finger movements in handling objects, and the difficulty in bringing each finger of the affected hand separately and accurately to the tip of the thumb have been described above, but these defects are even more apparent when the patient attempts to use simple and familiar tools. When a man with a right-sided cerebellar lesion was given a pair of scissors he had, in the first place, difficulty in grasping them correctly, then failed to direct them properly and was unable to move the blades regularly and appropriately when he attempted to cut a piece of paper with them.

In writing, too, these disturbances are very obvious when the wound involves the right half of the cerebellum. The pencil is held incorrectly and insecurely, grasped too tightly, and its point is pressed much too firmly on the paper. The letters are frequently unequal in size and irregularly spaced; the individual letters are badly formed and their lines are often jerky and angular. Further, as he writes it can be seen that he pauses frequently, especially between the up and down strokes, and the movements of his pencil are consequently interrupted and discontinuous. If he uses a pen he is liable to run its point through the paper and make ink splashes; his attempt with it is therefore less successful (fig. 10).

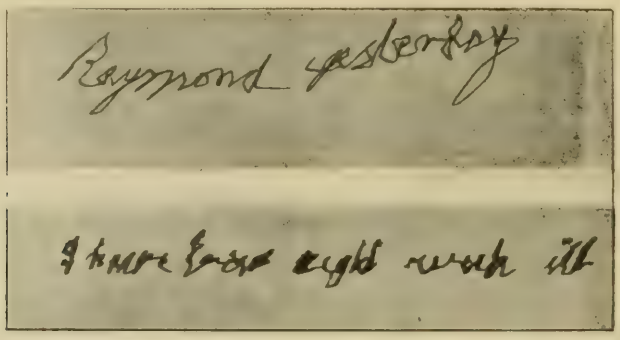


FIG. 10.—Specimens of the writing of two men with right-sided cerebellar injuries. The upper is of the words, "Raymond, yesterday"; the lower, "I have been eight weeks ill."

Static Tremor.

I have not observed tremor in any case while the limbs and body were at rest and fully supported, but it often appears during the maintenance of posture that requires muscular contractions. That which occurs during movement has been already described as a component of "cerebellar ataxy."

When a patient with a unilateral cerebellar lesion sits or stands there is frequently tremor of his head, which usually consists of fine or moderate irregular oscillations in any or all directions; it increases as he becomes tired. Similarly, when standing his whole body is often displaced by larger and more irregular swaying movements. This unsteadiness seems to be due to irregular and discontinuous contractions of the muscles that should maintain the attitude.

Tremor is as a rule seen in the affected limbs only when he strives to adopt a posture or as they tire, which they do more rapidly than the normal. When an attitude is once attained, as when his arms are outstretched horizontally in front of him, or his legs raised from the couch and flexed to right angles at the hip and knee, the affected limb is usually held for a

time as steady as its fellow, and often indeed even more so. It is noteworthy that the fine vibratory tremor which can often be seen in the normal arm, and even more distinctly felt, when it is held rigidly extended and unsupported, is generally absent in the affected limb; in one case its absence was so obvious that the photographer who exposed a plate as the patient held his arms in this position remarked how much steadier the affected one was. This condition corresponds to that which Babinski has described as *cerebellar catalepsy*, or the property of limbs remaining for a long time immobile under the influence of will; but otherwise I have seen no evidence of this symptom in the earlier stages of cerebellar lesions. On the contrary, when the limb begins to tire, and especially if it is still asthenic, a coarse tremor often develops, which is partly due to failure of sustained contraction of the fatigued muscles, but largely to the repeated voluntary attempts of the patient to bring it back to the position from which it tends to fall away. The tremor which occurs in these conditions is comparable to nystagmus and consists of two phases, namely, slow displacements by gravity and quicker voluntary jerks back towards the original position.

Before describing the abnormalities of standing and gait produced by unilateral cerebellar lesions, it is advisable to consider other symptoms which influence them. These are particularly the attitude assumed by the patient and the occurrence of vertigo.

Vertigo.

Many text-books include vertigo among the symptoms of cerebellar disease, but much doubt has been thrown on its direct relationship to cerebellar lesions; in fact, most authorities attribute it to affection of the labyrinth, or of the vestibular nerve or its nuclei.

Giddiness is an extremely common symptom after all gunshot injuries of the head, and almost all patients in whom the cerebellum is involved state on questioning that they were giddy at first or after regaining consciousness. From many of these no accurate description of the sensation could be obtained; but this is not surprising as most men are dull, obfuscated and unobservant for some time after being wounded in the head. Seventeen men, however, described apparent displacement of self, or of external objects, or of both. In twelve of these self and the external world seemed to be simultaneously displaced; in three there was apparent movement of self only, and in two solely of the external world. In fourteen of these seventeen cases the displacement took the form of rotation around the longitudinal axis of the patient's body; in two they were in the vertical plane; while one man stated that the walls of the room in which he lay seemed to be receding from him.

Among these fourteen patients the same difficulty was experienced in

attempting to determine definitely the direction of the rotation, since the vertigo rarely persisted more than one or two days. In almost all the subjective rotation of self and the movement of the outer world were in the same direction, but no constant relation could be discovered between the direction of the rotation and the site of the injury. In a series of cases of cerebellar tumour Dr. Grainger Stewart and I found that when vertigo occurred in patients with unilateral lesions the sense of movement of self and of external objects was from the side of the lesion towards the opposite side; but my present series, in as far as one can rely on the statements of the patients, does not confirm this rule.

It is interesting, however, that in the only three cases in which vertigo associated with actual movement occurred under observation, the patients rotated from the affected towards the opposite side (i.e., the shoulder of the affected side moved forwards) and the apparent movement of the external world was in the same direction. In the early stages of other cases too, there was a noticeable tendency for the patients to lie on the affected side. Some expressed considerable reluctance to rest in any other position, though one man stated that if he fell asleep lying on this side he was liable to roll over on his face. Consequently when "forced movements" were observed they took place in the same direction as they do in animals after ablation of one half of the cerebellum.

Further, the common attitude of a patient with a unilateral cerebellar injury, the head rotated towards the opposite side and the homolateral shoulder in advance of its fellow, may be looked upon as a latent tendency to rotation in this direction.

In the two cases in which during vertiginous attacks a sensation of vertical displacement only occurred, external objects seemed to rise in front of the patients, and one man complained that simultaneously his head seemed to be sinking back through the pillow. In both of these men the wound was over the middle of the posterior surface of the cerebellum, which was also much the most common site in the series as a whole.

Spontaneous Deviation of the Limbs and the Pointing Test

which Bárány has described, especially in connexion with labyrinthine vertigo, may be dealt with here.

In order to examine for spontaneous deviation of the upper limbs the patient is asked to hold both arms extended horizontally in front of him and close his eyes. It is advisable for the observer to steady his hands and check any tendency to movement till his eyes are closed. Then in the majority of cases with unilateral lesions the homolateral arm swings either slowly or abruptly away from the symmetrical position and comes to rest gradually. This occurred in all the thirty-three

cases in which it was looked for. In thirty-two the arm swung outwards, and in one slightly inwards, but the latter case, in which one lateral lobe of the cerebellum was almost completely destroyed and death occurred on the third day, may be neglected as the arm was so asthenic that it could scarcely be raised. We may consequently assume that the involuntary deviation of the unsupported arm is almost constantly outward.

While the observer's hands are steadying the patient's arms he can often feel a definite tendency to active displacement of the homolateral arm in this direction.

When in slight injuries this deviation is not obvious, it may be brought out by shaking or tapping the affected arm, this then generally moves in the direction towards which it tends to deviate.

Frequently the arm also deviates upwards or downwards from the level in which it was originally placed.

Barány's pointing test confirms these observations. The patient's extended forefinger is placed in contact with some fixed object, as the observer's finger, which is held at some distance above the bed, and he is then asked, while his eyes are closed, to bring his finger down to the bed and slowly up again to the object; with his normal arm he can reach the mark accurately or approximately on each attempt, but in most cases the forefinger of the affected side deviates constantly to one side of it, and this deviation increases for a time if the test is continued. In order to avoid conscious efforts to correct the error it is advisable for the observer to allow the moving finger to strike a similar object that can be moved into contact with it; if a tape measure is held horizontally in such a position that the patient's finger must touch it the error in each movement can be easily measured. If the more proximal segments of the limb are fixed the tendency to deviation of its distal segments can be investigated in the same manner. In the early stages of an extensive injury, the homolateral limbs are often so asthenic and ataxic that the test cannot be easily employed.

In all the thirty-two cases of unilateral lesions, involving different parts of the cerebellum, in which the arms were tested, a tendency to deviation was observed in the limbs of the affected side, and in all the deviation was outwards from the vertical when the arm was moved at the shoulder only. In certain cases vertical movements at the elbow and wrist were also examined, and in these, too, the deviation was constantly outwards, though the error was here much smaller.

In twenty-seven cases movements in the horizontal plane were similarly investigated; in nine progressive deviation upwards occurred, in seven the error was downwards, in six there was no tendency to deviation, and in the other five the direction of the error was irregular. The direction of the error in horizontal movements is not, however, always constant, in a few cases it was in one test upward and in another downward.

When this test is first seen these errors towards a fixed point in movements that are not controlled by vision suggest strongly a disturbance of the sense of position or of the appreciation of movement ("muscle sense"), in the affected limb, but it can be easily demonstrated that this does not exist; the patient can, for instance, while his eyes are still closed, bring his other forefinger accurately and promptly to the tip of that one which is actually several centimetres distant from the point he wishes to touch by it, showing that he is fully aware of its position in space. This can be confirmed by other tests too.

A tendency to deviation of the lower limbs is less common and the error is less pronounced when it exists. It is remarkable, however, that while the affected arm constantly, or almost constantly deviates outwards, the homolateral leg generally swings inwards (adducts), or is raised obliquely upwards and inwards over the opposite limb. In fifteen cases the pointing test was applied in vertical movements; in four there was no appreciable error, in two the leg deviated outwards, and in nine progressively inwards.

Another feature brought out by the pointing test is a remarkable tendency of the affected limb to overshoot the mark; while the normal limb is arrested at or near the position of the object which the patient wishes to touch, the affected one almost always swings past it, and often as much as 30° to 45° beyond it. This is obviously a manifestation of dysmetria; no disturbance of any form of sensation that could be responsible for it was demonstrable in any case.

Attitude.

Abnormal attitudes, which are such a prominent feature in animals after experimental lesions of the cerebellum, are much less constant and striking in man. Considerable importance has, however, been attached to the position of the head in cerebellar disease, especially when the affection is unilateral; in gunshot injuries its significance is generally doubtful, since the wound frequently involves the cranial attachments of the neck muscles, and because the patient naturally assumes the position which is most comfortable or which gives him least pain. As a rule, however, the head tends to be flexed towards the side of the wound and rotated towards the opposite side, so that the chin approaches the contralateral shoulder and the occiput is consequently approximated towards the shoulder of the affected side. This is particularly striking in cases in which the wound has healed.

Special attitudes of the trunk are less obvious, but in the early stages of an injury the body is often concave to the side of the lesion even as the patient lies on his back; this is usually more prominent when he sits up, but as he then inclines to and tends to fall towards this side the

upper part of his trunk is often purposely bent over in order to maintain equilibrium so that his spine is convex in this direction.

In certain patients, especially in those who are still or were recently subject to vertigo, the upper part of the trunk also tends to rotate towards the unaffected side (i.e., the homolateral shoulder is advanced), especially when they close their eyes; this attitude can be made more distinct by gently shaking the bed or the chair on which they sit.

In some of my patients, and particularly in those in whom the vermis cerebelli was also involved, the face was stolid and expressionless.

The flaccid and hypotonic limbs generally assume any posture into which they fall or are moved, or which may be given them by gravity, but when the patient holds the arms outstretched that homolateral to the wound is frequently more abducted than its fellow, and in several cases it is described in my notes as more rotated inwards at the shoulder and pronated at the elbow. In a few cases the hand of this side tended to be saddle-shaped, with the wrist flexed and the fingers hyperextended at the metacarpo-phalangeal joints, when the arms were held out.

There is no common noteworthy attitude of the lower limbs as the patient sits or lies in bed.

Standing and Gait.

When a patient who is able to leave bed is placed on his feet for the first time he is shaky, uncertain and unsteady, his whole body sways irregularly, his head oscillates, and he is usually in considerable danger of falling, especially towards the side of the wound and backwards. It often seems as if he were impelled to this side; several patients have, in fact, complained that they felt that they were pulled over, as though by an invisible hand, in this direction. It is, however, remarkable that even after being confined to bed for several weeks with a wound of one side of the cerebellum the patient can generally maintain his equilibrium, and make awkward though appropriate and often successful attempts to recover it if it is threatened. At this stage his attitude in standing is very striking; his head and trunk are both inclined to the injured side and his spine is concave to it, but his pelvis is so tilted that his weight falls chiefly on his opposite foot. The homolateral shoulder is generally higher than the other and it is almost always in front of it, and the trunk is occasionally rotated on its longitudinal axis towards the unaffected side. The homolateral leg is usually abducted and sometimes rotated outwards. His whole body is held stiff and rigid, and even when in danger of falling he does not sway it naturally in his efforts to save himself.

If he is gently pushed to either side, or if his shoulders are tapped firmly, it is found that he can be more easily thrown over towards the side of the lesion and that he makes less appropriate efforts to save him-

self when falling in this direction. Finally, he can usually stand on his unaffected leg alone, but when he attempts to place his weight on the homolateral limb, he sways and staggers so much that considerable support may be necessary to prevent him from falling. In cases of severe injury this leg also tends to give way suddenly under him as though it were too feeble to bear his weight.

It must be emphasized that the patient can stand as securely with his eyes closed as when they are open, there is consequently no tendency to Romberg's sign. On the other hand, he becomes more unsteady and is in greater danger of falling if his attention is diverted, even if only by conversation; he obviously supplements the normal muscle reflexes that preserve equilibrium by voluntary effort.

When such a patient attempts to walk many remarkable features are seen in his gait. If the lesion is severe and extensive he has often much difficulty in preserving his equilibrium and is frequently in danger of falling, though almost all men with unilateral lesions succeeded in walking unaided on the first day they left bed; this varied between eight and seventy-one days after the infliction of the wound.

The first and most striking feature is the patient's obvious fear of trusting himself on the affected leg. He throws his weight on it slowly and cautiously, and at each step hurries off it by bringing the other limb forward and to the floor, as quickly as he can. At the same time he stumbles, and tends to fall to this side. His steps are generally short, unequal in length, and irregular. In advancing the affected leg, the foot is occasionally dragged along the ground, but more frequently it is raised unnaturally by excessive flexion of his knee and hip, and is brought down with undue force. Further, it often comes irregularly to the floor, the whole sole falling on it simultaneously, or the toes before the heel; and, occasionally, either the inner or the outer margin of the foot alone may first come in contact with it.

This leg is usually abducted and rotated somewhat outwards, but this is not constant; often, in fact, the homolateral foot is brought in front of the other in one step, and in the next is abducted too much.

When the patient is able to walk safely alone, he still stumbles to the homolateral side, and reels abruptly from his proper course in this direction; in walking between two rows of beds, he is therefore liable to run into that on his affected side. He consequently takes a zig-zag course, the abrupt deviations being always towards the side of the lesion, except when he stumbles awkwardly, as he often does, towards the other side in an effort to save himself, or to correct the deviation. The stumbling is often due to excessive adduction of the affected limb, owing to which its fellow in being advanced trips on it; but it is most commonly a manifestation of the tendency to fall to the affected side.

In walking along a straight line he deviates, or shows a tendency to deviate, towards the affected side. This is usually more obvious when his eyes are closed; then his course is often a parabolic curve concave to the side of the lesion. His attitude, with his head rotated towards the opposite side, the homolateral shoulder in advance of its fellow, and the transverse axis of his body consequently oblique, often gives the impression that his direction, even when he walks straight, inclines to the homolateral side of the line he intends to follow.

This tendency to deviation can be well demonstrated by making the patient walk round a chair or a small table; when the shoulder of his injured side is inward, he frequently runs into the chair, while he constantly deviates outwards from it, so that he traces an opening spiral, in walking round it in the opposite direction. In walking backwards, on the other hand, especially when his eyes are closed, he generally deviates in a sharp curve concave towards his affected side.

The patient soon becomes aware of this tendency to stumble, reel, and deviate towards his affected side, and attempts to control it; occasionally, indeed, in his efforts to prevent falling to the one side, he actually stumbles to the other. These symptoms are usually most marked when he walks quickly; but a few patients got along better rapidly than slowly and deliberately.

Further, when walking quickly, he has difficulty in stopping suddenly; when ordered to halt, he often cannot pull up for two or three steps; or, in attempting to do so, he may throw himself forwards so abruptly on to his toes that he is in danger of falling on his face. In certain cases the difficulty in stopping when walking backwards was even greater—one man, for instance, on taking a step or two backwards, could not pull himself up till he ran into some object, or was assisted, for several weeks after the infliction of the wound. This difficulty in stopping resembles that seen in paralysis agitans, in so far as it is due to the legs having to move after the body in order to keep the centre of gravity above the base of support; but in cerebellar lesions it must be attributed to deficient synergia of the trunk and thigh muscles by which this should be promptly attained. Another example of asynergia is furnished by the fact that as the patient walks the affected arm hangs inertly by his side, and does not swing synchronously with the movements of the opposite leg; nor does he use this arm naturally in attempting to balance himself. Further, when he is in danger of falling, he does not move either his trunk or arms naturally or adequately in attempting to regain equilibrium, and if he is suddenly pushed in any direction he falls stiffly and rigidly like a doll.

In cases of unilateral cerebellar lesion the chief abnormalities in gait are therefore due to the irregular and incorrect placing of the affected foot, and to the tendency to fall, stumble, and deviate towards the affected

side. The patient soon attempts to correct these disturbances by voluntary effort; he throws his weight as little as possible on the unsafe leg, and when he can control his direction by vision, he checks, though rarely completely, his tendency to deviate from the direction he wishes to follow.

The disturbances in gait are, it is true, usually greater in relation to the disorders of movement of the affected leg, when this is tested as he lies on his back, than might be expected; but the symptoms of cerebellar deficiency are always most obvious in the most complicated actions, and more complex synergias, involving both the trunk and limb muscles, are called into play in maintaining equilibrium and direction during gait than are required in movements of the leg alone. Many of the older physiologists and certain clinicians have regarded the cerebellum as an organ concerned mainly with the maintenance of equilibrium; but this hypothesis finds no support in the examination of men in whom one side of the organ only has been damaged; in fact, the successful efforts to maintain equilibrium, despite the patient's deficient control over the movements of the one leg, are often surprising.

When a man who has recently left bed attempts to progress on his hands and knees, similar disturbances are seen. The affected arm is abducted, this shoulder is held lower than the other, his trunk is concave to the side of the injury, the hip of this side is depressed, and the leg is usually somewhat adducted. In moving forwards, the affected arm is raised too high, and the hand swoops down too heavily to the ground; the homolateral knee, on the other hand, is generally dragged along the floor. The patient occasionally falls to the side of the lesion, but more frequently he merely slides over to this hip, owing to its excessive adduction, or to the leg giving way under him. The similarity both in attitude and progression on the hands and knees between a patient with a unilateral destruction of the cerebellum and a dog or monkey after ablation of one half of this organ, is consequently very striking.

Disturbances of the Ocular Movements, and Nystagmus.

In animals subjected to cerebellar injuries disturbances of the ocular movements have been observed, but little emphasis has been laid on the occurrence of nystagmus; this is not surprising, as it can be properly investigated only on voluntary movements and fixation of the eyes, and with the co-operation of the subject. Many physiologists have even refused to admit that the nystagmus is dependent on the cerebellar lesion (Munk, Rothmann). It is however remarkable that even from most clinicians these symptoms have not received the attention they deserve; the reason probably is that they are particularly prominent only early after acute lesions.

For some days after a unilateral gunshot wound of the cerebellum the eyes while at rest are generally deviated towards the opposite side, especially

if the patient happens to be unconscious, and at first it is often difficult to make him move them conjugately towards the injured side. When he attempts this the range of movement is occasionally incomplete, but usually a more striking feature is its slowness and the effort necessary to execute it. In one patient at least in whom it was carefully investigated this conjugate paresis was associated with erroneous projection towards the homolateral side. The paresis diminishes gradually but the difficulty in movement to this side, in comparison with deviation in the opposite direction, frequently persists for weeks. The vertical movements of the eyes and convergence are never similarly affected.

In five cases the position known as "skew-deviation" was observed, that is, the homolateral eye was directed downwards and inwards while the other looked upwards and outwards. This lack of parallelism in the optic axes disappeared however on fixation when this could be obtained, and consequently diplopia did not result. Skew-deviation was observed only during the first week or so after an injury of or an operation on the cerebellum, except in two cases in which rapid destruction or compression of this organ, owing to abscess formation occurred.

Nystagmus is a much more common symptom; it was present in fact in almost every case in which injury to the cerebellum produced any trace of functional disturbance, and its chief characters are so constant that it must be regarded as a clinical symptom of the highest importance. It is essentially a fixation nystagmus, that is, it can be seen as a rule only when the patient fixes an object.

It is advisable to describe it first as it most commonly occurs in the early stages of a severe unilateral lesion. In such a case some nystagmoid jerks can be usually observed when the patient fixes an object in any part of his visual field, except in the region of that point, usually 10° to 30° to the unaffected side of the middle line, towards which his eyes when at rest tend to deviate. We shall speak of this as the "rest point."

When he looks at an object held directly in front of him his eyes tend to deviate slowly towards the unaffected side and are brought back to the middle line by sharp jerks of small or moderate range. These movements are usually slow in rate and at first regular, but when the patient's effort or attention begins to tire his eyes may move more widely before their deviation is checked, and the correcting jerks consequently become less regular in rate and larger in range.

It is on looking towards the injured side however that the nystagmus is most pronounced, then it consists of wide, slow deviations towards the middle line, or more correctly towards the "rest point," and forcible jerks of large amplitude, slow in rate and fairly regular in rhythm, towards the side to which the eyes are voluntarily directed. Its rate was estimated in several patients and in all varied between twenty-three and

thirty oscillations in ten seconds. Both movements are as a rule strictly horizontal, but occasionally slight rotation, generally downwards, may be seen in the quick phase. This nystagmus generally remains regular in rate and amplitude for a short time, but as the patient tires his eyes are less frequently and less completely pulled back towards the point he should fix.

In this stage too there is almost always nystagmus on fixing an object to his unaffected side, but it is more rapid, finer in range and less regular. Here too the slow deviation is towards the "rest point" and the movements are most commonly horizontal. Frequently in looking towards this side the nystagmus consists of series of rapid jerks with short pauses between each series.

Nystagmus is less constant and less regular in vertical movements. In the majority of early cases, however, the eyes on being directed upwards tend to deviate slowly towards the horizontal and are brought back by sharp quick jerks of small range, which are rarely quite regular in rate or well sustained. Almost always there is either some rotation of both eyes upwards and towards the side of the injury in the quick phase, or the quick movement is more or less obliquely upwards and to this side. This occurs especially when the point fixed is brought to the affected side of the mesial sagittal line. On looking downwards regular and sustained nystagmus is rare, but very commonly slow deviations towards the horizontal line occur, which are corrected by quick, relatively small jerks vertically downwards, or more frequently obliquely downwards and to the side of the lesion, or with rotation to this side. The nystagmus that occurs on vertical movement always increases, or it may appear only when the object fixed is brought to the homolateral side of the middle line.

Finally, on convergence both eyes often tend to deviate away from the side of the lesion and are brought back to their proper position by irregular jerks of small range.

Unless there is much paresis of conjugate deviation towards the injured side the movements are more regular, larger in range and better sustained the farther the point fixed is from the primary central position, or rather from the "rest point," and the jerks increase in range and accelerate in rate when the point is moved farther from this position. The nystagmus also tends to become less regular and less rapid as fixation tires, but it can be evoked again by renewal of fixation, especially if the object fixed is moved slightly farther from the middle line.

This is the typical form of nystagmus produced by recent cerebellar injury, but divergences from it are common. Little or no disturbance may be, for instance, seen on movement of the eyes in one direction, though characteristic nystagmus is associated with all other movements; or the relative amount that occurs when the eyes are moved in different

planes may not be typical. These divergences may be partly dependent on the localization of the cerebellar lesion, though I am not yet able to state definitely that this is so. It varies, however, according to the severity of the injury; when this is recent and extensive and the eyes deviate spontaneously towards the unaffected side and movement to the other is difficult, no nystagmus, or only an occasional jerk, may occur when the patient looks to that side, and on fixing to the side of the lesion only a few slow irregular jerks of large range can be seen before the effort at fixation becomes ineffective.

If the lesion is slighter, or some time has elapsed since the infliction of the wound, and there is no obvious deviation of the eyes, and at the most only slight difficulty in conjugate movement towards the injured side, the nystagmus is also less regular and less characteristic. On central fixation the eyes generally remain steady, or only an occasional deviation of small range towards the unaffected side occurs, which is at once corrected by a sharp jerk. On looking towards the side injured a slow, coarse, forcible nystagmus, with its slow phase towards the rest point, still develops, but it is usually less regular and less well sustained. After a time the oscillations become more rapid and smaller in range before the true nystagmus disappears or is replaced by occasional irregular jerks of the eyes. On fixing to the opposite side the excursions of the eyeballs are still smaller in range and more rapid in rate, but they are less regular and persist for a shorter time. Vertical movements are now less commonly complicated by nystagmoid jerks, and these are in fact rare on downward deviation. In a few severe cases there was at this stage more rotation upwards and to the side of the lesion when the patients looked directly upwards, though in one man, in whom the rotation had been previously to that side, it was upwards and in the opposite direction four weeks after the injury was received. Here, however, the mastoid process and the internal ear probably, too, was damaged.

Finally, in cases with very slight injury, or at a later period after the infliction of a severe wound, the only abnormality in the movements of the eyes may be occasional irregular slow deviations towards the middle line and sharp rapid jerks, irregular in rate and in amplitude in the opposite direction, on prolonged fixation towards the injured side; or there may be only slight irregularity of the eyeballs when they are fully deviated in this direction.

It is obvious that the nystagmus produced by a unilateral cerebellar lesion is essentially a fixation nystagmus, that is, it is seen chiefly when an attempt is made by the patient to fix accurately a point in any part of his visual field, but it may also occur when accurate fixation is impossible. In some of the cases spectacles with high convex lenses, such as have been employed by Wilson and Pike, were placed in front of the patient's eyes,

and it was then found that when he moved them to order the oscillations were considerably less marked, or did not occur on deviation in certain directions.

Nystagmus of cerebellar origin is therefore characterized by the facts that it occurs chiefly in fixation, that the slow phase is always towards the primary central position or the rest point, and that it is more constant, more regular, better sustained, and the oscillations of the eyeballs are slower in rate but larger in amplitude, when the patient looks towards his injured side. Finally, it persists for weeks, or even months, after the onset of the lesion, though it gradually becomes less regular, or is replaced by irregular nystagmoid jerkings.

The question as to how far this nystagmus is dependent on co-existing labyrinthine lesions at once arises, more especially since labyrinthine disturbances, as Moutier and others have shown, commonly result from gunshot injuries of the head. It cannot be denied that the internal ear was possibly damaged in certain cases of my series, and particularly in those in which the missile entered through or near the mastoid process, but in the majority it certainly escaped, and no essential difference in the type of the nystagmus could be detected in the two groups. Further, the nystagmus has not the character of that produced by destructive or irritative labyrinthine lesions. We owe valuable contributions on this subject to Gordon Wilson and Pike, and to the former I am indebted for the following distinguishing features of the nystagmus which results from destruction of one labyrinth: it persists at the most only two or three days; it is increased when fixation is cut out by suitable lenses placed in front of the eyes in a spectacle frame or by other means, and the slow phase is always towards the injured side, whether the eyes are at rest or deviated voluntarily towards either side.

If the features of that produced by cerebellar lesions are compared with these, it will be seen that nystagmus of cerebellar origin differs in many important particulars from that due to destructive labyrinthine lesions, and we may consequently conclude that injury of the labyrinth plays no direct part in its pathogenesis.

The nystagmus associated with cerebellar lesions has also been attributed to injury or disease of neighbouring centres, and especially to lesions of Deiters' and Bechterew's nuclei. This factor can be certainly excluded in the majority of my cases, and the regularity with which the characteristic nystagmus occurs, even when the wound is small and relatively superficial, and in the absence of all signs of medullary and pontine involvement, makes it extremely probable that it is due to damage of the cerebellum alone.

(*To be continued.*)

MALARIA IN MACEDONIA WITH ESPECIAL REFERENCE TO THE USE OF PROPHYLACTIC QUININE.

By Captain C. H. TREADGOLD.
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Between the beginning of February and the end of April, 1917, I had occasion to examine the various units of a certain Division in Macedonia with a view to determining the proportion of men *on duty* who were infected with malaria. Smears from 540 men were stained and searched for parasites, and the results are summarized in Table I. Plasmodia were found in over thirty per cent of the men who had been in Macedonia during the previous summer, while the altered differential leucocyte count suggested that over sixty per cent of such men were in reality infected. Had it been possible to reckon the absentees in the Malta and Salonika hospitals, the total percentage of infected men would, of course, have been much greater.

It is interesting to note that parasites were found in nearly eight per cent of men with no history of fever; if we regard the increased hyaline count as evidence of infection, their number increases to over thirty-seven per cent.

No evidence of infection being found in recent drafts, it may be assumed that the great majority if not all the men in whom parasites were present had been infected during 1916 or earlier.

Technicalities.—The average time spent over each slide was about ten minutes, and no man's blood was examined more than once. Gametes were found in over ninety per cent of the positive slides, and schizonts were also present in a large proportion. *Plasmodium vivax* was the common parasite found; *P. falciparum* was noticed on seven occasions, *P. vivax* being also present in four out of the seven; *P. malariæ* was only diagnosed once. To simplify matters, *P. falciparum* and *P. malariæ* have been reckoned in the table as *P. vivax*.

The Results of Quinine Prophylaxis.[1]

Method of Administration.—The salt employed was the sulphate, the daily dose (five to thirty grains) being given either in acid solution or in the form of tablets.

Results of Analysis.—Chemical analysis showed the drug to be pure.

Results of Administration.—The men in Groups I, II and III of Table I had been taking quinine during the greater part of 1916, and I was assured by the regimental medical officers that many were taking it at the time I made the blood films. The material summarized in Table II indicates the

[1] By quinine prophylaxis is meant the regular administration of quinine with a view to preventing the development or recrudescence of clinical malaria.

kind of evidence available. These twelve men belonged to the same brigade, the majority coming from a regiment whose medical officer had had considerable experience of malaria in the East, and who was positive that quinine was being ingested in the doses stated. The persistent slight deafness which was such a characteristic feature in half of them would seem to corroborate his statement.

TABLE I.

Groups	Total examined	*P. vivax* present	Hyaline leucocytes : 15 per cent or over in men in whom no parasites were found
I.—Macedonia, during summer of 1916 ; fever within three months of examination	300	120 (40 per cent)	89, thus increasing percentage of presumably infected men to 69·6
II.—Macedonia, during summer of 1916; fever during this period but none within three months of examination	93	16 (17·2 ,,)	33, thus increasing percentage of presumably infected men to 52·6
III.—Macedonia, during summer of 1916 ; no history of fever	63	5 (7·9 ,,)	20, thus increasing percentage of presumably infected men to 39·6
IV.—Drafts reaching Macedonia between September 1, 1916, and December 31, 1916 ; never out of Britain before ; no history of fever	40	3 (7·5 ,,)	11, thus increasing percentage of presumably infected men to 35·0
V.—Drafts reaching Macedonia in 1917 ; never out of Britain before ; no history of fever	44		

TABLE II.

No. of case	Cinchonism at time of blood examination	Vivax, schizonts and gametes in peripheral circulation	Daily dose of quinine	Length of time since last relapse
1	—	Yes	30 gr. daily for weeks	1 month
2	Tinnitus and deafness, especially on left side	,,	,, ,, ,,	3 weeks
3	Slight deafness	,,	,, ,, ,,	2 ,,
4	—	,,	20 gr. daily for weeks	3 months
5	Tinnitus and slight deafness	,,	10 gr. daily for weeks	3 ,,
6	—	,,	,, ,, ,,	2 ,,
7	—	,,	,, ,, ,,	2 ,,
8	Slight deafness	,,	,, ,, ,,	5 weeks
9	Tinnitus and slight deafness	,,	,, ,, ,,	2 ,,
10	—	,,	,, ,, ,,	1 week
11	Slight deafness	,,	,, ,, ,,	8 days
12	—	,,	,, ,, ,,	1 month

Owing to the varying amounts of quinine ordered by the medical officers attached to the different regiments, it was possible to place 125 out of the 150 men examined during the month of February into one or other of the following groups (*vide* Table III). Whether the quinine was given in

small or large doses or not at all, seemed to make little difference either to the number of relapses or to the proportion of cases in which parasites were found; many of these men were constantly relapsing.

TABLE III.

Groups	Number of cases examined	Number of men in whom parasites were found
I. No quinine between attacks ..	26 ..	16 (61·5 per cent)
II. 30 gr. daily	21 ..	11 (50·2 ,,)
III. 20 ,, ,, ...	9 ..	6 (66·6 ,,)
IV. 15 ,, ,, ...	6 ..	4 (66·6 ,,)
V. 10 ,, ,, ...	48 ..	22 (45·8 ,,)
VI. 15-30 gr. bi-weekly	11 ..	3 (27·2 ,,)
VII. 6 gr. daily and 12 gr. bi-weekly ..	4 ..	3 (75·0 ,,)

The evidence so far considered indicates :—

(1) That despite infection, a fair proportion of the men examined had —temporarily at any rate—resisted illness or got the upper hand of the disease.

(2) That a great deal of sickness due to malaria nevertheless existed in the Division, much of it being little influenced by the administration of quinine.

The figures available from French sources show an even higher degree of infection, but entirely confirm these observations. For example Garin,[2] although he gives no details as to the number of men examined, claims that plasmodia—usually gametes—can be found in the blood of from sixty to eighty per cent of troops returning from Macedonia without a history of malaria, while Abrami[3] states that from eighty-five to ninety-five per cent of the French Macedonian Army was attacked by malaria in spite of early and general preventive quininisation. An early and correct solution of the following problems is therefore urgently needed :—

I.—Is any appreciable proportion of the Macedonian Force saved illness through the administration of prophylactic quinine? If so, what proportion?

II.—Is the general course of the disease ever affected by the previous administration of prophylactic quinine? If so, is the sum of such influences favourable or the reverse?

To answer these questions as they should be answered clearly demands a very exact knowledge of the conditions upon which the usefulness of the drug, both as a prophylactic and as a curative agent, depend. Unfortunately these conditions have never been thoroughly investigated on scientific lines and large gaps in our knowledge consequently exist. In spite of these drawbacks I have been tempted to analyse and draw conclusions from the data available in the literature of the subject.

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[2] Garin, *Presse Médicale*, June 4, 1917.

[3] Abrami, P., *Presse Médicale*, March 22, 1917.

I.—*Is any appreciable proportion of the Macedonian Force saved illness through the administration of prophylactic quinine? If so, what proportion?*

The question will be considered under the following headings:—

(*a*) General considerations followed by an analysis of the available statistical evidence.

(*b*) The different methods of giving prophylactic quinine.

(*c*) The rôle of suggestion in quinine prophylaxis.

(*d*) Conclusions.

(*a*) General Considerations followed by an Analysis of the Available Statistical Evidence.

Altogether I had access to some two hundred original articles, reports of medical societies, etc., in which the question of quinine prophylaxis was either dealt with or discussed. Nearly all of them were written in English, French or German, and all—with one exception—were published between the discovery of the malaria parasite in 1880 and the present time. 52·2 per cent of authors gave no details of their personal experience, while only 16·9 per cent made use of controls. Table IV gives an idea of the opinions expressed by these different writers, no less than eighty per cent of whom were in favour of quinine prophylaxis.

TABLE IV.

	In favour of quinine	In favour of quinine with reservations	Against	Total
No statistics	65	21	20	106
Statistics but no controls ..	43	6	12	61
Statistics with controls ..	26	0	8	34
Total ..	134	27	40	201

The majority of these papers were, however, devoid of interest. No doubt a large number of writers were unfavourably influenced by the difficulties in the way of controlling their results and realized the impossibility of appreciably increasing our knowledge in the absence of such controls. Be this as it may, the difficulties in the way of experimental research have unfortunately brought extremists to the fore, and such statements as the following are scattered throughout the literature. "The whole trend of recent inquiry and the results of clinical experiment have been to show that at the present day more than ever before, the scientific administration of quinine is established as a measure of very great value and that it is indispensable in the general prevention of the disease."[4] "Quinine has its position in relief and cure, but in sanitary administration it has none."[5]

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[4] Brown, W. Carnegie, *Trans. Soc. Trop. Med. and Hyg.*, London, iv, p. 193.

[5] Macdonald, A., *Trans. Soc. Trop. Med. and Hyg.*, London, x, p. 1.

In such controversial subjects as the one under discussion, when extreme views are expressed in the absence of adequate evidence, the truth usually lies between the two extremes. Knowing as we do that resistance to malarial disease is considerably greater in the inhabitants of malarial countries than it is in immigrants from non-malarial ones, it would seem natural to suppose that, other things being equal, *the benefit derived from the use of prophylactic quinine varies directly as the resistance of the individual taking it*—in other words, the greater the resistance the better the statistics.

The truth of this theory can only be tested in the present state of our knowledge by analysing the statistical evidence from different countries, taking care to differentiate between the natives of such countries and immigrants.

This has been accordingly done in Tables V to VIII.

Analysis of Statistical Evidence from 1880 *to Present Time.*

TABLE V.—Countries Bordering on Mediterranean Basin.

	In favour of quinine	In favour of quinine with reservations	Against	Total
Statistics but no controls ..	16	0	1	17
Statistics with controls	18	0	0	18
	—	—	—	—
Total ..	34	0	1	35

All thirty-four papers in favour of quinine prophylaxis deal with the question in so far as it affects the permanent inhabitants of the respective countries; the single paper against deals with temporary inhabitants, viz., the French Army in Macedonia.

TABLE VI.—Africa, Excluding the Mediterranean Area.

	In favour of quinine	In favour of quinine with reservations	Against	Total
Statistics but no controls ..	13	1	3	17
Statistics with controls	2	0	2	4
	—	—	—	—
	15	1	5	21

One of the four papers in which controls were used deals with natives and is in favour of quinine prophylaxis; the remaining three deal with immigrant whites—one paper being in favour and two against.

TABLE VII.—Southern Asia.

	In favour of quinine	In favour of quinine with reservations	Against	Total
Statistics but no controls ..	14	1	6	21
Statistics with controls	5	0	4	9
	—	—	—	—
	19	1	10	30

Three out of the nine papers in which controls were used deal with natives and are in favour of quinine prophylaxis. The remaining six deal

either with immigrants or with partly immigrant populations such as coolies on rubber plantations; two were in favour of quinine prophylaxis, four against.

TABLE VIII.—North and South America, including West Indies.

	In favour of quinine	In favour of quinine with reservations	Against	Total
Statistics but no controls	3	3	1	7
Statistics with controls	1	0	2	3
Total ..	4	3	3	10

One out of the three papers in which controls were used deals with natives and is in favour of quinine prophylaxis; the other two deal with immigrants and are against.

A study of these tables reveals the existence of a state of affairs very different to what might have been anticipated from merely glancing over the opinions of the various writers summarized in Table IV, especially when we remember that *controlled results are the only results of any scientific value.* When controls were used and natives of the respective countries investigated, no less than twenty-three or 100 per cent of workers were in favour of prophylactic quinine. When controls were used and immigrants dealt with, only three out of eleven or 27·2 per cent were in its favour. The evidence available would therefore seem to justify the enunciation of the following law: "*Other things being equal, the natives of malarial districts derive more benefit from quinine prophylaxis than immigrants from non-malarial ones.*"

Let us now attempt to estimate the real value of quinine prophylaxis to the natives of such districts.

An attempt to estimate the real value of prophylactic quinine to the natives of malarial countries in the Mediterranean Area.—We have seen that all the available statistical evidence is in favour of administering prophylactic quinine to the inhabitants of Mediterranean districts in which malaria is endemic. What does this really amount to? Celli, one of the best known of the Italian workers, thus expresses himself: "The daily use of quinine is as necessary as daily bread during certain months."[6] "He who takes quinine every day and therefore has always a supply of quinine in the blood-stream, can undergo with impunity inoculations of blood full of malarial parasites and can expose himself with little or no danger to the bites of infected mosquitoes."[7] One might almost conclude from this that the Italian with a little quinine in his pocket was in a position to snap his fingers at malaria under the most unfavourable conditions. Yet a little later the following analysis of very extensive clinical material was published by the same author[8]:—

[6] Celli, A., *Journ. Trop. Med.*, London, 1908, xi, 101.

[7] Celli, cited by Henson, "Malaria," Henry Kimpton, 1913, 136.

[8] Celli, cited by Stuart (M.), *Proc. Canal Zone. Med. Assoc.* for the half year, 1910, 67.

N.B.—This Table is reproduced by several authors.

TABLE IX. (TAKEN FROM CELLI).

Mosquito prophylaxis + quinine prophylaxis ..	Symptoms in 1·75 per cent of cases	or 1 in	57
Mosquito prophylaxis only	,, 2·5 ,,	,, 1 ,,	40
Quinine prophylaxis only	,, 20 ,,	,, 1 ,,	5
No protection	,, 33 ,,	,, 1 ,,	3

Clearly the quinine given produced some effect, but compared with the results obtained by mosquito prophylaxis, its action was almost negligible.

The brothers Sergent, who have investigated the subject for many years in Algeria, seem less enthusiastic than they used to be. The last paper I have seen gives details of the quinine given to native children in 1910.[9] One of their tables is reproduced, and the authors content themselves with the observation that all methods give appreciable results:

TABLE X.—TAKEN FROM SERGENT (E. AND É.).

Dose of quinine	Improvement	No change	Worse
0·2 grm. daily	19 (5·5 per cent) ..	315 (83·5 per cent) ..	7 (2·02 per cent)
0·6 ,, every three days	19 (4·2 ,,) ..	425 (94·6 ,,) ..	4 (0·9 ,,)
0·6 ,, every six days ..	35 (9·7 ,,) ..	300 (80·4 ,,) ..	16 (4·8 ,,)
Controls (no quinine) ..	5 (0·8 ,,) ..	220 (90·5 ,,) ..	21 (8·7 ,,)

These Italian and North African results are not very impressive, especially when it be remembered that we are dealing with the only type of case in which a consideration of the general statistical evidence led us to suppose that really striking benefit would be uniformly attained, viz., natives who have presumably inherited and acquired a certain amount of immunity against the parasites of their own country.

(*b*) THE DIFFERENT METHODS OF GIVING PROPHYLACTIC QUININE.

The following are the two methods usually employed :—

(1) A small daily dose—five grains, for example.

(2) A larger quantity—usually fifteen grains, taken on two consecutive days at intervals of from a week to ten days.

The advocates of method No. 1 claim that quinine taken in this way is always circulating in the blood, and object to No. 2 because it leaves the individual defenceless for several days owing to the rapid excretion of the quinine absorbed.

The partisans of method No. 2 maintain that by giving quinine in this manner the curative effect of the drug is not interfered with, but that the use of No. 1 may lead to the production of quinine-proof strains of parasites. I have searched the literature but can find no evidence of one method having any appreciable advantage over the other.

It is interesting to note that in Italy, Greece, and North Africa, three

[9] Sergent, E. and É., *Ann. de l'Inst. Pasteur*, Paris, 1913, xxvii, p. 372.

to six grains daily is the ordinary dose employed, and larger quantities are not usually supposed to give any better results. On referring to Table III we notice that, on the whole, the smallest percentage of cases was diagnosed in those who were taking least quinine. The possible significance of these facts will be considered later.

(*c*) The Rôle of Suggestion in Quinine Prophylaxis.

In quinine prophylaxis, as in all forms of medical treatment, the personality of the physician is not without influence, while the extent of his confidence in the treatment proposed can usually be gauged by the patient with considerable accuracy. These considerations explain to some extent the ephemeral success of many new drugs and methods of treatment. Traces of a similar process can be found in the history of quinine prophylaxis. Laveran,[10] for example, in his book on malaria, published in 1898, comments on the fact that the English, who previously wrote so much in favour of prophylactic quinine, seem to have got tired of it.

The psychological aspect of malaria prophylaxis has received considerable attention from American writers, several of whom regard the co-operation of the public as essential, and agree that no means of stimulating its curiosity and of keeping its sympathy should be neglected.[11 and 12]

In Macedonia the French authorities advertise the merits of quinine by means of humorous coloured posters and picture post cards, which must go far towards reconciling the "poilu" to his daily dose. In Algeria the brothers Sergent have initiated the use of pink tablets containing three parts of sugar and two of quinine bihydrochloride, and state that these are everywhere accepted by the natives with the greatest alacrity, in marked contradistinction to their attitude towards the classical method.

These things are really worth bearing in mind, and the striking possibilities of suggestion are vividly brought home to one by the following incident, related in all seriousness by Henrot.[13] "Dr. Nouat, head of the Military Hospital, Calcutta, was consulted by the Governor of Bengal as to the best way of protecting messengers against malaria. So many died from pernicious malaria as a result of the unhealthy country they had to cross, that messengers had become unobtainable. Dr. Nouat, acting on a theory prevalent at the time—that infection took place via the respiratory tract—constructed respirators containing finely divided animal charcoal, and made the messengers wear them; the effect was excellent, for the messengers, although taking exactly the same route as their predecessors, ceased being ill."

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[10] Laveran, *Traité du Paludisme*, 1898.

[11] Henson, G. E., *New York Med. Journ.*, 1910, xcii, p. 724.

[12] Craig, C. F., *South Med. Journ.*, Nashville, 1911, iv, pp. 469, 478.

[13] Henrot, H., *Bull. Acad. de Méd.*, Paris, 1895, xxxiv, pp. 292, 325.

(*d*) Conclusions as to the Effect of Quinine in Preventing Clinical Malaria in Macedonia.

(1) *Primary Malaria.*—We saw in Table I that a proportion of the men exposed to the risk of infection escaped, evidently because—(*a*) They had never been bitten by infected mosquitoes, or (*b*) if infected, their powers of resistance were equal to the occasion and no clinical symptoms ensued.

To what extent had the natural resistance of these latter been reinforced by taking quinine?

Although the drug must have been the decisive factor in a few cases, the previously considered evidence points to their number having been very limited. No matter what disease we take, a proportion of any population is always more or less refractory. Moreover, malaria may be a very chronic condition, and the presence of parasites in the blood of people who have never developed symptoms, and who have never taken quinine, is perfectly well known. As Bass[14] says: "No doubt many people get infected without ever knowing anything about it; others infected under similar conditions may be ill and in some cases die." Further, the absence of symptoms in such apyrexial carriers is no guarantee of permanent immunity; in many cases all that is needed to produce an attack is such slight lowering of vitality as is produced for example by a chill, a surgical operation, or a broken bone.

(2) *Recurrent Malaria.*—I have come across no evidence which leads me to suppose that quinine is any more efficient in preventing relapse than it is in aborting the initial attack (*vide* Tables II and III).

II.—*Is the general course of the disease ever affected by previous quinine prophylaxis? If so, is the sum of such influences favourable or the reverse?*

The matter will be discussed under the following headings:—

(*a*) The clinical evidence and its significance.

(*b*) The mode of action of quinine in malaria both as a prophylactic and as a curative agent.

(*c*) Conclusions.

(*a*) The Clinical Evidence and its Significance.

The following examples are taken from the literature of the subject:—

(1) Celli states that both doctors and patients are unanimous that daily prophylactic quinine very frequently aborts primary infections, notably diminishes relapses, and prevents cachexia.[15]

(2) So far back as the American Civil War the responsible medical authorities[16] state that men were saved from attack and preserved in perfect health for the time being by the use of prophylactic quinine, but

[14] Bass, C. C., *Kentucky Med. Journ.*, Bowling Green, 1917, p. 393.

[15] Celli, A., *Journ. Trop. Med.* London, 1908, xi, p. 101.

[16] "The Medical and Surgical History of the War of the Rebellion," 1888, iii, 165.

that the method was abandoned owing to the shortage of quinine and to the eventual sickness of such men.

(3) Neiva (Brazil) who was in charge of 3,000 men in a highly malarial district found that to obtain the same therapeutic effect, the dose of quinine had to be increased in proportion to the length of exposure to infection.[17]

(4) Bell (Panama) states that men who were receiving the drug, in comparison with those who were not, under otherwise identical conditions, furnished fewer cases in the proportion of three to four: that such men were, however, more refractory to treatment, had a more prolonged convalescence and relapsed more frequently.[18]

(5) Stitt[19] is of the opinion that quinine in reasonable doses has little or no effect, while those who take it are less amenable to its curative action and relapse more frequently than those who only take the drug at the time of an attack; he also maintains that quinine prophylaxis does not prevent either anæmia or splenic enlargement, while Jacobson[20] goes so far as to say that although symptoms may be masked for a time, the actual damage is as great as though no quinine had been given.

(6) We have seen that in Macedonia quinine alone is quite unable to prevent malaria, while very large doses have to be given in many cases to obtain clinical amelioration. Only a few weeks ago at a meeting of the Society of Tropical Medicine and Hygiene, a paper was read by Sir Ronald Ross in which details were given of various intensive forms of quinine treatment in chronic cases of malaria invalided from Macedonia. No matter how big the dose or what route was chosen for its administration, frequent relapses still occurred.

Comment: After studying this and similar evidence, we are bound to admit that the general course of the disease may be adversely influenced by the previous taking of quinine. How is such a thing possible and why do different observers get such remarkably discordant results? The answer to the last question will be considered first.

Why does Quinine give such Variable Clinical Results? — Under ordinary circumstances the key to the problem undoubtedly lies in the constitution of the patient and in his state of health both at the time of infection and subsequently. We know that under good social conditions intermittent malaria tends to cure itself both temporarily and permanently, and that whether quinine be given or not, the temperature returns to normal in a few days. If only a little outside help is required to change the balance in favour of the patient, good hygienic conditions, an enthusi-

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[17] Neiva. Cited by Hudleston, Journal of the Royal Army Medical Corps, London, 1913, xxi, 320.

[18] Bell. Cited by McGuire, *U.S. Nav. Med. Bull.*, 1914, viii, p. 571.

[19] Stitt, E. R., *Journ. Amer. Med. Assoc.*, Chicago, 1908, i, 1682.

[20] Jacobson, A. C., *New York Med. Journ.*, 1906, lxxxiv, p. 1224.

astic physician, and a little quinine will suffice either to definitely cure the individual, or at any rate to prevent his relapsing for a considerable time in the absence of reinfection. If on the other hand the resistance of the patient falls short of this, no marked benefit will result.

We have just seen that clinical evidence favours the view that the course of malaria may be unfavourably affected by the previous taking of quinine. How can this occur and what is the mechanism of the process?

Discussion of the Probable Mechanism.—We have seen that under relatively favourable conditions a small daily dose of quinine tends to prevent clinical malaria or at all events to lengthen the incubation period. This, however, cuts both ways, and should an individual run the risk of being frequently infected, the tendency will be for him to receive more massive doses of sporozoites as the result of taking prophylactic quinine. Can we wonder if his malaria is of a severer type when it does develop? It is universally assumed that the sporozoites resulting from conjugation in the insect host possess a greater potential capacity for mischief in the vertebrate host than merozoites derived from many generations of asexual multiplication. We know that a European in the Tropics who stays out at night and is generally careless with regard to mosquito prophylaxis is much more likely to suffer from and to be eventually invalided home with chronic malaria than the individual who is careful in these respects. Even experimental evidence is not altogether wanting, for experiments on proteosoma infections in sparrows are said to show that the severity of the disease largely depends on the dose, that is to say on the number of sporozoites inoculated.[21]

We are now in a position to appreciate the inadvisability of giving prophylactic quinine over long periods, and to realize that the severity of malaria in Macedonia has been undoubtedly increased in some cases by previous quinine prophylaxis.

(*b*) The Probable Mode of Action of Quinine in Malaria.

(1) *As a Prophylactic.*—"Quinine differs from most other important alkaloids in acting not on some specialized form of living matter, but on the general nutrition of almost all forms of protoplasm. Experimental evidence indicates that a number of ferments act more vigorously in very dilute solutions of quinine, while their action is retarded by larger quantities."[22] It is possible, therefore, that the lytic action of human plasma on malarial parasites is increased when quinine is present in suitable concentration. It is equally possible that too great a concentration of quinine retards this activity; also that the long continued administration of the drug sets up a tolerance to its presence which necessitates an increase of concentration to get the original effect.

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[21] Gill, C. A., *Indian Journ. Med. Research*, 1914, ii, p. 268.

[22] Cushny, "Pharmacology and Therapeutics," 1915.

We have previously noted that certain doctors in Mediterranean countries regard 0·2 gramme (about three grains) daily as sufficient; also that the few cases I had the opportunity of examining in Macedonia appeared to justify the view that a maximum daily dose for prophylactic quinine exists and that it is not a very big one. It is also possible that the mechanism about to be considered is not without influence.

(2) *The Action of Quinine as a Curative Agent.*—Barlow, as the result of experimental work, thinks that quinine in therapeutic doses does not directly destroy the plasmodium, but tends to poison infected red blood corpuscles in such a way that they are more easily acted upon by the excretory products of the parasite. This results in its liberation before segmentation has taken place and facilitates its destruction. Gametes escape because their less active metabolism does not liberate sufficient toxin to destroy even the quinine-poisoned cell.[23]

Comment.—This explanation is in harmony with the facts. We know, for example, that quinine given at the beginning of an attack of intermittent malaria, does not appreciably influence the train of clinical events but tends to prevent subsequent attacks. It would seem to do this by poisoning infected red corpuscles and accelerating their disintegration, thus making the conditions less favourable for the multiplication of the parasite, which is unlikely to be present in sufficient numbers to produce symptoms when the next attack is due. The functioning of this mechanism would evidently give the resisting powers of the individual time to rally. Should his vitality be insufficient to dispose of all the plasmodia the metabolism of the survivors may be assumed to slow down, and even very large doses of quinine may be unable to poison the red cells sufficiently for the parasitic toxins to destroy them with sufficient speed, the plasmodium reacting by slowing its metabolism still further. In many cases it successfully avoids extermination, and in the event of the host's vitality being sufficiently lowered a relapse occurs.

In severe and chronic cases resulting either from massive infection or from the feeble resisting power of the individual or from a combination of the two, large doses of quinine are usually given over long periods; according to the above theory the poor results obtained would be largely due to an increased tolerance of the red cells for quinine.

(*c*) Conclusions.

Both clinical and theoretical considerations compel us to admit that the general course of the disease may be noticeably affected by the previous taking of quinine, and that the sum of such influences is frequently unfavourable. So far as the Allied Armies in Macedonia are concerned, there is every reason to suppose that quinine taken daily over

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[23] Barlow, *Amer. Journ. Trop. Dis.* and *Prev. Med.*, 1915, ii, p. 585.

periods of many months has increased the severity and chronicity of the disease in a certain proportion of cases. Taking one thing with another the available evidence indicates that, in Macedonia at any rate, the disadvantages of quinine prophylaxis outweigh the advantages.

General Summary of Conclusions.

(1) Small doses of prophylactic quinine, not too long continued, are of proved utility to the natives of malarial countries, both in the absence of anti-mosquito measures, and when such measures are incomplete.

(2) Quinine may be given with advantage to immigrants under the following conditions: (*a*) During short journeys when conditions with regard to mosquito prophylaxis are less favourable than usual. (*b*) An occasional dose after an unusually tiring day. (*c*) Nervous people may be advised to take an occasional course as an additional precaution in spite of efficient mosquito prophylaxis.

Speaking generally, it may be stated that quinine prophylaxis is usually a bad investment for immigrants in the absence of protection against mosquito bites; taking one thing with another, the practice would in their case seem to be little more than a pious fraud which has been perpetuated from one generation to the next, simply because public opinion throughout the world has never been sufficiently enlightened to encourage the working out of the problem on scientific lines. A few well planned experiments, carefully and simultaneously conducted in several countries, and these conclusions might have been reached many years ago. We know the experimental method to be at the bottom of all scientific and industrial progress, yet we refuse to admit its very obvious utility in Preventive Medicine. The following observation applies only too literally to the subject under discussion: "En dehors des sujets dont le contrôle expérimentale est facile, bien peu d'opinions s'appuient sur la logique rationnelle. Sur l'interprétation de faits au fond assez clairs, mais que n'étayent pas des expériences suffisament tangibles, le désaccord est complet."[24] Surely this lamentable state of affairs cannot continue much longer? The experimental evidence exists, but unfortunately its neglected fragments lie embedded in a very extensive literature, much of which is of little value owing to lack of detail and to the impressions of the writers being uncontrolled by clinical experiment.

APPENDIX.

Although the difficulties in the way of efficient mosquito prophylaxis in non-immune armies operating in highly malarial countries are undoubtedly very great, the failure of quinine prophylaxis shows that more serious efforts will have to be made in order to overcome them. A few methods of procedure are therefore mentioned and their value briefly discussed.

[24] Le Bon, C., "Premières Conséquences de la Guerre," 1916.

A.—The Destruction of Mosquito Larvæ.

(1) *Subsoil Drainage.*—Everyone agrees that this is the ideal method. Unfortunately finance and labour difficulties militate against its general adoption. In Panama, where both money and labour were available, this method was little used, owing to the engineering works constantly interfering with the drains.[25]

(2) *The Oiling of Surface Water.*—In Panama, the system of oiling seems to have been perfect, the whole area for about half a mile round all dwelling places being oiled once a week so thoroughly that all mosquito larvæ were killed.[25] The method is obviously a good one when sufficient oil can be obtained.

(3) *The Piping of Streams.*—In ravine country when mosquitoes breed in the pools associated with fast running but temporary streams, such streams may be piped with advantage within half a mile of camps.

B.—Mechanical Prophylaxis Against Mosquito Bites.

In countries where the reduction of mosquito larvæ is difficult, the importance of mechanical prophylaxis can hardly be exaggerated. The question will be briefly considered under the following headings.

(1) *The Screening of Dwellings.*—In the Canal Zone, all houses occupied by Americans had double doors, the windows being protected with copper-bronze screens, eighteen mesh to the inch. Crenstein[26] concludes that properly screened dwellings alone can be depended upon to reduce by at least one-third the malaria incidence in a locality where malaria is endemic.

(2) *Net Prophylaxis.*—Stott[27] and others have emphasized the great value of supervised net prophylaxis for soldiers.

(3) *Veils and Gloves with Gauntlets.*—These have been employed on sentry duty, etc. If properly used such methods should be fairly effective, but adequate supervision is obviously difficult or impossible.

(4) *Essential Oils.*—Oil of citronella, etc., either pure or in the form of ointment, has been tried for men on night duty, but without much success. According to Celli and others such methods have a very restricted action.

(5) The employment of African or Greek troops for sentry duty and all forms of night work during the summer and autumn months would seem to be worthy of serious consideration. After going carefully into the matter, the advantages might be found to outweigh the disadvantages.

(6) The last but certainly not least important method is the education of officers and men as to the habits of the mosquito, and the means of avoiding infection. Pamphlets, posters, lectures, lantern slides, cinema films, all have their place according to circumstance and opportunity.

In conclusion I should like to thank my brother, Captain H. A. Treadgold, R.A.M.C., for valuable assistance.

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[26] Crenstein, A. J., *Journ. Amer. Med. Assoc.*, Chicago, 1914, lxiii, 1931.

[27] Stott, H., *Indian Med. Gaz.*, Calcutta, 1915, i, p. 382.

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(2) The names of workers who attempted to control their results are marked with an asterisk.

(3) All reference to literature which merely expresses the personal opinion of the author without giving details of his work is omitted.

"MYALGIA"—ITS DIAGNOSIS IN FORWARD AREAS.

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Introductory.

The term "myalgia," originally introduced in order to rescue a certain class of case from the heading "rheumatism," shows a tendency under the conditions of diagnosis which obtain in forward areas to be expanded so as to hold almost as much as the term "rheumatism" itself.

In view of the importance of accurate diagnosis in these cases, both from the point of view of treatment, and from the point of view of economy in man power—that a man may be allocated early to work suited to his physical powers—inquiry was made to ascertain the extent and the directions in which it might be possible to improve the diagnosis "myalgia" (as generally made) by the application of simple and rapid criteria suitable to the difficulties which attend diagnosis in the front line.

And further, inquiry was made into the leading symptoms of myalgia as seen by the Regimental Medical Officer, in order to provide a basis on which, after the exclusion of conditions likely to be confounded with it, the diagnosis "myalgia" might rapidly and reasonably be made, and (an important point) the severity of the case adjudicated.

The importance of accurate diagnosis at the outset of a patient's journey towards rear areas is enhanced by the great weight which any diagnosis once made carries with it: and the reluctance of a Medical Officer in a rear area to change the diagnosis—forgetful of the fact that it may have been made in a shell hole. And another reason why the diagnosis "myalgia" is seldom corrected is that myalgia, by reason of the intangibility of its symptoms and the opportunity it affords to malingerers, has got a bad name, and is generally regarded as a nuisance rather than as a problem.

The observations (an account of which follows) were conducted at a field ambulance over a period of three months, and with a front line battalion over a period of twelve months. A few cases necessary to complete the series were seen at a casualty clearing station.

I.—Differential Diagnosis.

The application of the rough and rapid diagnostic criteria shown below to 200 cases reaching field ambulance and casualty clearing station diagnosed "myalgia" resulted in an improvement of the diagnosis in 60 cases excluding those newly diagnosed "neuralgia," and in 108 cases including the new diagnosis "neuralgia" (i.e., in more than one half), as shown in the following table:—

Pyrexial Conditions.—(P.O.U.O., etc.) (19 cases 9·5 per cent). In almost all cases the pyrexia was in the evening only. It is noticeable, especially in winter and when for any reason it is difficult to keep the

troops warm, that a patient may have an evening temperature as high as 102° F., and yet be subnormal at the sick parade early next morning. Hence the pyrexial nature of the condition is easily overlooked, unless there is an unofficial evening sick parade or some such arrangement.

TABLE SHOWING THE RESULT OF AN ATTEMPT TO IMPROVE THE DIAGNOSIS IN 200 CASES DIAGNOSED "MYALGIA."

New diagnosis	Criteria applied	Number of cases
Pyrexial conditions	Pyrexia (usually evening only)	19
Post-pyrexial conditions:	History of "P.O.U.O.," or trench fever, within previous five weeks with more or less continuous pain since	—
Neuralgia*	Pain, aggravated when patient is lying down, warm, and perfectly still	12
Ostalgia*	Ditto; tenderness over shins	5
Unspecified	Pain on movement; stiffness aggravated after rest	2
		— 19
Arthritis:	Creaking	0
Osteo-arthritis	Palpable bony deformity	3
Rheumatoid arthritis	Peri-articular infiltration	6
Unspecified		7
		— 16
Sciatica	Kernig's sign; nerve tenderness	4
Neuralgia†	Pain, aggravated when patient is lying down, warm, and perfectly motionless	46
Muscular stiffness following considerable exertion	History	2
Gout	Joint signs	1
Muscular cramps	History	1
	Total	108

* Usually accompanied by pain on movement.

† Previous history of "P.O.U.O.," or "trench fever," in twenty-one cases, occurring from five weeks to one year previously.

Post-pyrexial Conditions.—(19 cases = 9·5 per cent). In the majority of cases the patient was able to state that he had been in hospital a short time (within five weeks) previously and that "it said P.O.U.O. on the card." (Patients appear to take a most painstaking interest in their cards). In a few cases only the pyrexial nature of the forerunning illness had to be judged by the patient's history of his symptoms—headache, sweating, etc.

The predominant features of these post-pyrexial pains are: (1) That they are present in an aggravated degree when the patient is warm in bed and lying motionless; and (2) that they are referred to the shin bones in many cases, there being in a few cases actual tenderness of the subcutaneous surface of the tibia. True myalgic pains, in marked contrast to this, are absent at complete rest but aggravated after rest, until the stiffness which has increased during rest has worked off.

Pyrexial and post-pyrexial conditions (nineteen cases each) thus account for thirty-eight of the 108 cases in which the diagnosis "myalgia" was susceptible of improvement. The importance of the alteration is seen

as soon as the question of treatment is raised; the treatment of myalgia by massage, liniments, Turkish baths, etc., being definitely contra-indicated in pyrexial conditions, and hardly the best for post-pyrexial neuralgia and ostalgia in which delayed convalescence seems to be the main factor.

Arthritis.—(16 cases = 8 per cent) (osteo-arthritis 1·5 per cent, rheumatoid arthritis 3 per cent, unspecified 3·5 per cent). The joints affected were mainly knees. A few shoulders were included in the series and one hip. The preponderance of knees is probably largely due to easy diagnosis. There is no doubt that a skilled diagnostician, especially if unhampered by time, would be able to take many more cases—especially shoulders, hips and spines—out of the myalgic abyss.

The figures are considerable enough in themselves, and become more so in view of the fact that the patients were drawn almost exclusively from what in Germany would be termed a "shock" division, and included few above forty years of age. The arthritis figures for labour battalions and so on would naturally be very much higher.

The importance of detecting the cases is economical. No treatment for "myalgia" makes them fit for winter work in the trenches. They are a burden to field ambulances and to hospitals, and are only a paper asset to their Division. But once the condition is recognized there is some chance that suitable employment may be found for the case.

Sciatica.—(4 cases = 2 per cent). The diagnosis is important from the point of view of treatment; adequate treatment of recurring sciatica being unobtainable at a field ambulance. Occasionally the rough diagnosis "sciatica" is made where "myalgia" would be nearer the mark.

Neuralgia.—(46 cases = 23 per cent). The importance of this distinction is at present mainly scientific. Patients generally classed as myalgics fall broadly into two distinct classes. In the one class there is no pain when the patient is warm in bed unless he attempts to move. Rest makes him stiff. This stiffness wears off with gentle movement. There is pain, often described as acute and stabbing, on quick movement. Prolonged exertion precipitates or increases the condition. This is true myalgia. In the other class there is pain of a character which is variously described as "aching," "gnawing" and "throbbing," which is *aggravated* when the patient is warm in bed and persists although he be absolutely motionless. He complains of inability to sleep. There is often considerable cutaneous hyperæsthesia. This class of case I have called "neuralgia." It is very distinct from myalgia.

The forty-six cases include two cases of severe neuralgia of the feet following exposure, and five cases more or less confined to the upper extremity. But the majority followed the usual myalgic distribution—viz., lumbo-sacral and lower extremity.

The curiosities of *muscular stiffness following considerable exertion* (2 cases = 1 per cent), *gout* (1 case = 0·5 per cent) and *muscular cramps* (1 case = 0·5 per cent) complete the 108 cases in which, in a series of 200, the diagnosis myalgia was susceptible of improvement by the application

of diagnostic criteria suitable, by reason of their simplicity, to forward areas.

II.—Symptoms and Signs of Myalgia.

After the exclusion of the conditions dealt with above under the heading "Differential Diagnosis," there remains a residue. A number of cases with rheumatic symptoms reporting sick at the medical inspection room of a battalion were examined with a view to determining what are the leading features of myalgia (using the term in a restricted sense) as seen in forward areas. The hope was entertained that a sufficient definition of the condition might be obtained to render diagnosis more certain and rapid.

The question of *malingering* assumed considerable importance in the inquiry. Gross malingering was in my experience uncommon; but the exaggeration of symptoms was very prevalent among those who were in the habit of reporting sick. A lugubrious outlook is common among patients, due as much to their failure to rise above the misery and hardship of their surroundings as to their actual pains. Such patients, whose spirit has been damped, even if not actually extinguished, form the considerable majority I think of the sick of the Army in forward areas; and are clearly unsuited to be the media of a scientific inquiry into which symptoms enter more than physical signs. Nor under war conditions may much tempering of the wind be accorded to these "shorn lambs"—at the expense of their more spirited comrades.

Only patients whose mental health had not suffered too severely from the strain of war are represented in the figures which follow. Thus the inquiry was begun in 264 cases. But in 214 of these it was abandoned either owing to another diagnosis being made, or owing to the vitiated mental outlook or inferior intelligence of the patient, or owing to the slightness of the case. The inquiry was concluded in fifty cases, which may be taken as typical of genuine uncomplicated myalgia as seen in forward areas.

The regions affected were as follows: Cervical 1, dorsal 9, intercostal 2, lumbar 36, sacral 28, shoulder 6, arm 1, elbow 1, forearm 1, wrist 0, hand 0, buttock 15; thigh: anterior 7, lateral 23, posterior 34, mesial 10; knee 31; leg: anterior 16, posterior 12, lateral 21; ankle 4, foot 6. An attempt to define more exactly the muscle groups involved by observing the effects of active and passive movement of various groups had to be abandoned owing to the claims of other work.

The distribution is usually symmetrical (thirty-six cases) and is in the majority of cases lumbo-sacral extending down the lower extremities for a varying distance. The lumbar and sacral pains are by no means always co-existent. In fact, the general rule is for the pain to be either lumbar *or* sacral, the thigh pains being superadded in each case. Occasionally the pains are present in the lower extremities but absent in the lumbar and sacral regions; and the converse is common in convalescence. One case of "stiff neck" occurred in the series and two cases of pleurodynia. No

case of pure acute lumbago was seen. In one case the pain was confined to the upper extremity.

Pain is absent when the patient is lying down, warm, and perfectly still (44 cases). This is a marked distinction from pyrexial, post-pyrexial, and neuralgic conditions. In these the pain is aggravated when the patient is warm in bed.

Rest makes the patient stiffer (46). This stiffness wears off on moving about gently (34). Active contraction and passive hyperextension, especially if quick, cause pain which is described as sharp rather than dull (45). Cold and damp weather precipitates or aggravates the condition (37). Prolonged exertion (route marching with full pack, digging, &c.) have a like effect (41). Massage is painful (27 cases out of 31), but beneficial (21 cases out of 31). The condition is chronic (previous attacks in 44 cases). The site of the pain is generally identical in successive attacks (29 cases out of 44). The onset is usually sudden (35).

There appears to be no connection between previous pyrexial attacks and myalgia. But in the case of neuralgia (see "Differential Diagnosis" above) there would seem to be a definite relationship. The young are not exempt (one case aged 19), but the condition is uncommon under 30, and increasingly common thereafter.

III.—Definition of Myalgia.

The practical delimitation of myalgia by more accurate differential diagnosis is, perhaps, more important than its definition in words. But from the foregoing it may be said that myalgia is a chronic condition, precipitated or exacerbated by cold, damp and fatigue, affecting groups of muscles, and characterized by muscular stiffness becoming greater on resting and less with movement; and by pain, absent at complete rest, and sharp on active contraction and passive hyperextension. It is to be distinguished from neuralgia by the facts that in neuralgia the pain is aggravated when the patient is warm and motionless, and that in neuralgia hyperæsthesia of the skin is commonly present.

IV.—A Method found Useful in Forward Areas for Examining Patients Complaining of Rheumatic Pains.

The number of cases of rheumatic pains coming under the notice of the regimental medical officer is large. In my experience he is called upon to give a decision in such cases upwards of 2,000 times in the course of a year in the forward area, the same patient often, of course, reporting sick repeatedly.

Under these circumstances the plan of making a fairly complete examination at the outset and, as an aid to memory, noting the result in an alphabetical book kept for the purpose, or by hieroglyphic in the patient's pay book, has been found to meet the case. It not only saves a vast amount of time in the long run, but provides at once a diagnosis that is as accurate as possible. It prevents the sick parade from becoming a farce, and ensures that justice is done alike to the genuine case and to the "skrimshanker."

The method of examination which follows is necessarily a simple and a rapid one, and takes more notice of symptoms than of physical signs; but it has been found useful in arriving at a fairly accurate diagnosis under conditions which make diagnosis difficult. The method is as follows:—

(1) Take the temperature, evening if possible. If not, pay special attention to headache and other feverish symptoms. While taking the temperature, note (2) general appearance; (3) apparent age; (4) pupils (especially for the contraction of insomnia); (5) conjunctival anæmia; (6) pulse; (7) respiration. Note also (8) the condition of the tongue.

These observations eliminate pyrexial conditions and acute illness generally.

Interrogate the patient as follows: (9) "Where do you get pain?" (10) "Where is it worst?" (11) "When do you notice the pain most?" (12) "Is there any pain when you are lying down, warm, and *perfectly still?*"

The answers to these questions may suggest arthritis (examine for creaking and gross joint signs), sciatica (examine for Kernig's sign and nerve tenderness), tibial ostalgia (examine for tenderness over the subcutaneous surface), or neuralgia (examine for cutaneous hyperæsthesia, and inquire for history of previous pyrexia).

Then proceed: (13) "Is this your first attack?" (14) "How long have you been subject to these attacks?" (15) "What brings them on?" (16) "Do they always affect you in the same place?" (17) "What do you do for them?"

The last question aids in determining the severity of the case.

The attempt should next be made to produce pain by active and passive movement, the patient's appearance being noted the while.

Then ask: (18) "Is the pain sharp or dull?" If he has had any rubbing or massage, ask (19) "Is massage painful?" (20) "Does it do you good?" Proceed to inquire about stiffness by asking: (21) "Does rest make the stiffness greater or less?" (22) "Does the stiffness get greater or less when you are up and moving about?"

Conclude with some such phrase as: (23) "When you move *quickly* you get a sharp stabbing pain. Can you do as much as anybody else if you go slow?"

The last question is a valuable one for testing the patient's mental outlook. It puts him on his mettle. A genuine case answers in the affirmative, or with a negative that is obviously reluctant. Cases in which there is unwillingness of the spirit as well as weakness of the flesh are not ashamed to give a negative, the tone of which is whining in "war-worn" cases, and impressive, indignant or sanctimonious in patients who are knowingly exaggerating their symptoms.

If the answers seem to coincide with the definition of myalgia given above, the diagnosis is made and a note registered for future reference as to the degree of the condition, and as to the degree, if any, of exaggeration employed.

POST-DYSENTERIC DIARRHŒA AND ITS TREATMENT.

An Excerpt from Notes on 2,000 Cases at the Military Dysentery Convalescent Hospital at Barton-on-Sea.

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This is the commonest sequela of an attack of acute dysentery, and it follows both the amœbic and bacillary form of the disease with little distinction between. The post-amœbic diarrhœa is certainly more persistent, and is more prone to relapses—the post-bacillary more easily controlled, and does not relapse so often as the former.

Ordinary astringents may abate the diarrhœa in both cases, and in some cases, no doubt, stop it, but in most it returns as soon as the astringent medicine is discontinued, and the case may go on like this for months.

The unsatisfactory results from the use of astringents led me to inspect regularly the dejecta, and much information was obtainable as to why astringents fail so frequently. The motions varied from very pale, and almost jaundice-like colour, to dark-brown green; they were full of undigested food particles, and were all more or less putrid and highly offensive.

The patients passing these motions, which vary in number from two to eight or ten daily, are naturally more or less debilitated and down in weight—poor appetites, sclerotics, yellow, dull, and injected, usually pale of complexion, languid and dull.

All the symptoms, objective and subjective, indicate a derangement of the liver functions—particularly a dyscrasia of the bile, a reduced output, or both.

A chemically deranged bile must needs result in a non-digestion of the bowel contents, and if also the bile flow is diminished, the antiseptic qualities being also inhibited, putrefaction of the bowel contents is directly encouraged.

No worse condition could exist where intestinal catarrh is present than that the contents of the already irritated intestines should be in such irritating and septic conditions, as already has been pointed out.

Astringents, therefore, often fail entirely, or only partially succeed in bottling up the poisonous bowel contents—and, perhaps, it is often fortunate they do fail.

Castor oil will give great relief to these cases, but only temporarily, so also will mag. sulph.; but neither will bring about a large flow of clean healthy bile, the thing so much required. This, however, can be easily done by full doses of tincture of rhubarb, and my routine is to give every such

case not less than from four drachms, or even six drachms, at one dose. Forty-eight hours having been allowed to work it off, the motions are again inspected. If their colour and composition are still unsatisfactory, a second, or even a third similar dose is given. When their colour assumes a healthy brown or yellow tone, it indicates that the whole bowel is now freely flushed with clean healthy bile, and its putrid contents got rid of. In many cases the diarrhœa now stops of its own accord, but if it still continues, a few doses of some binding medicine will be now safe and successful. Usually I give thirty grains bismuth subnitrate with ten drops hydrochloric acid and ten of tinct. opii, two or three doses daily.

The diet should be soft and bland so far as possible, and a tonic containing pepsine should follow cessation of the diarrhœa.

Calomel is too severe, and causes a lot of colic; so does podophyllin. Euonymin is uncertain. Small doses of tinct. rhei are quite useless; the dose must be large, and its action full and speedy.

In Carrier cases tinct. rhei co. is useless.

Clinical and other Notes.

VINCENT'S ANGINA.

By Captain E. EMRYS-ROBERTS, M.D.
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In view of the widespread incidence amongst our troops on this front of affections of the throat and gums due to the fusiform bacillus and spirochæte of Vincent's angina, it may be of value to indicate a method of treatment that has met with unvarying success since its adoption in the area served by this laboratory during the last eighteen months. The treatment consists in the local application of the following lotion :—

Hydrogen peroxide	℥v
Vinum ipecac.	℥iii
Glycerine	℥v
Aq. ad	℥viii

By its use the familiar gingivitis is usually cured in about six days, while the throat condition clears up altogether in from twenty-four to forty-eight hours.

Vincent's angina of the throat is not only met with as a separate infection, but is very frequently associated with diphtheria. An analysis of the cases that have reached this laboratory shows that in nearly sixty per cent of diphtheritic throats both fusiform bacilli and spirochætes are present in a further fifteen per cent.

The rationale of the formula was based upon the following considerations: Firstly, that hydrogen peroxide is especially useful in loosening and clearing up purulent exudates, and, moreover, would militate against the growth and multiplication of the anaerobic fusiform bacillus; secondly, that vinum ipecacuanha might prove to have a more or less specific action upon the spirochæte; and thirdly, that glycerine by virtue of its hygroscopic and penetrative properties would effectually convey the preceding ingredients into otherwise inaccessible recesses. Whatever may be the theoretical value of these surmises, the practical issue is beyond doubt, and the consistent results obtained would appear to merit the general use of this lotion in Vincent's angina infections.

I wish to thank Captain Mallet, L.D.S., Captain Place, L.D.S., and the many medical officers in casualty clearing stations and field ambulances who have kindly reported their results.

VENEREAL DISEASE IN THE FIELD.

By Captain F. CROSBIE.
Royal Army Medical Corps.

The purpose of this article is to outline briefly the methods which have proved most satisfactory in dealing with venereal disease in a hospital in the field, where one is working entirely under canvas and with a necessarily restricted armamentarium.

The results may be roughly gauged from the statistics appended to various portions of the article, these having been compiled from the admissions and

discharges during the year 1916. They do not include sixty-nine officers treated during the year. Each of the recognized varieties will be dealt with in turn: (1) gonorrhœa; (2) venereal sore; and (3) syphilis.

Gonorrhœa.

On admission of a suspected case a film is taken from the urethra and microscoped immediately. It is only in chronic cases that a diagnosis is made from the clinical history alone, as a large proportion of these fail to yield gonococci on direct examination.

Having made a definite diagnosis in an early case one proceeds to treatment, and this again is controlled as far as possible by the microscope.

In the following description the expression "negative film" is used to denote one in which both gonococci and pus are completely absent.

(1) Urethro-vesical irrigations with 1 in 8,000 potassium permanganate solution three times a day. The man is taught to do this in the erect position, and in the great majority of cases no difficulty is experienced. The apparatus consists of a four-gallon petrol tin, suspended from a marquee pole at a height of ten feet from the ground; this is connected with a metal Miocchi nozzle by six feet of rubber tubing. We have twelve of these outfits in constant use, and it takes an average of four minutes for each irrigation.

(2) When the man has successfully accomplished the above for about five days, protargol (two drachms of a five per cent solution) is instilled along his entire urethra by means of an olivary catheter attached to a Guyon's syringe. This is performed immediately after the mid-day irrigation three times a week.

(3) Massage of the glands in relation to the urethra (viz., prostate, Cowper and seminal vesicles) is instituted about the third or fourth week—i.e., when the discharge has become watery and gonococci have disappeared.

(4) As soon as films prepared subsequent to the above procedure prove "negative," and all pain is absent, dilatation with curved metal sounds is resorted to.

(5) If in spite of this films remain "negative" all treatment is discontinued for three days, and a film taken before micturition on the fourth morning.

(6) If this is "negative" silver nitrate (two drachms of a one per cent solution) is instilled, and films examined for gonococci on three successive mornings. If these do not appear, the patient is discharged.

In confirmed cases the strength of the potassium permanganate solution may be gradually increased up to 1 in 2,000, or 1 in 200 zinc sulphate substituted. Where a mixed infection is present, and oxycyanide of mercury is unobtainable, solutions of hydrarg. biniodide may be employed in strengths varying from 1 in 40,000 to 1 in 10,000.

In chronic or relapsing cases treatment from the beginning should combine all stages up to and including "D." If one is not in possession of a dilator of the type of Kollmans, dilatation should be pushed at least up to 20 English.

Cases are grouped as follows:—

(*a*) Fresh: Those in which the discharge has been constant since infection, and therefore not necessarily at an early stage.

(*b*) Uncertain: Those which have had a previous attack which has cleared up, and have been exposed to reinfection recently.

(*c*) Relapse: Those in which all symptoms had been cleared up, and in which discharge recurred without any admitted exposure to infection.

	Total Per cent	Fresh cases Per cent	Uncertain cases Per cent	Relapses Per cent
Number of cases treated ..	802	473	102	199
Average number of weeks in hospital	9·23	9·01	8·22	9·04
Percentage of cases relapsing after discharge from hospital ..	3·49	2·32	1·96	7·53
Percentage of cases admitted with complications	15·83	13·31	11·76	25·62
Percentage of cases developing complications	3·49	3·38	2·94	3·01
Percentage of cases evacuated to hospital ship	1·87	0·84	0·90	5·02
Complications.				
Percentage of cases admitted with epididymitis	9·22	9·09	8·82	11·05
Percentage of cases developing epididymitis after first admission..	2·99	3·81	1·96	2·01
Percentage of cases admitted with arthritis	1·99	0·84	0·90	5·52
Percentage of cases developing arthritis after first admission..	0·49	0·21	0·90	1·00
Percentage of cases admitted with condylomata acuminata.. ..	3·24	3·59	2·94	5·02
Percentage of cases developing condylomata acuminata after first admission	—	—	—	
Percentage of cases admitted with stricture	0·99	0·63	—	2.51
Percentage of cases developing stricture after first admission ..	—	—	—	—

Venereal Sore.

Venereal sores may be classified according to their bacteriological origin as follows:—

(A) Those due to the spirochæte of syphilis.

(B) Those due to the strepto-bacillus of Ducrey.

(C) Those due to ordinary "septic" micro-organisms.

The clinical picture presented by an uncomplicated case from any one of these three groups is fairly constant. Unfortunately, for purposes of diagnosis, one frequently finds a combination of two, or even of all three, varieties in the same lesion. For this reason alone one should never state that any particular sore is definitely non-syphilitic. Neglect of this rule may lead to results beneficial neither to the welfare of the patient nor the reputation of the surgeon.

As soon as a man with a sore is admitted, simple hot water fomentations are ordered. The following day, films of the exudate are stained with weak Giemsa's solution and examined for the spirochæte of syphilis. Should the latter not be present, and a few weeks have elapsed since the man was exposed to infection, a sample of blood-serum is sent to the laboratory and submitted to the Wassermann reaction. In the event of a negative result being returned this may be repeated if considered necessary, but every effort is made to avoid retaining a a man in hospital merely for diagnostic purposes.

The treatment of Group A cases is obvious.

Group B cases yield most readily to the iodine derivatives, while those in Group C usually require preliminary fomenting, followed by the application of some weak mercuric preparation.

X-rays have proved useful in intractable cases, excepting those which subsequently proved to be syphilitic.

Syphilis.

The routine treatment administered here covers a period of five weeks, and comprises :—

A. Intravenous injection of "kharsivan." Five doses of 0·3 gramme are given at weekly intervals. In order to avoid serious reactions I would insist on five cardinal points :—

(1) Patient to be prepared as for a general anæsthetic, and complete rest ordered for at least twelve hours after injection.

(2) Freshly distilled water to be employed in the preparation of all solutions used.

(3) Each 0·3 gramme "kharsivan" (after neutralization) to be diluted with normal saline to at least 150 cubic centimetres before injection, and injected within fifteen minutes of preparation.

(4) Perfect clarity of the injected solution.

(5) Fluid injected not to enter blood-stream at a lower temperature than the latter.

Total number of intravenous injections of "kharsivan"—2,021. Of these 75 per cent were followed by temperatures of over 100° F.; 7 per cent were followed by vomiting; 31 per cent were followed by rigors; 70 per cent were followed by headache; two cases developed marked urticarial rashes.

B. Daily inunctions with mercuric ointment. The ointment used is composed of ung. hydrarg. fort. one part, vaseline three parts, and one drachm of this is inuncted daily. Close supervision is exercised to insure that this is correctly performed, as I regard it as the most essential part of the treatment, and that to which the patients evince the greatest dislike.

C. Intramuscular injection of mercurial cream. Ten minims (equivalent to one grain) are injected at weekly intervals over a period of five weeks.

As far as possible the patients suffering from venereal sores or syphilis are encouraged to attend to dental and oral hygiene. The rapidity with which salivation, etc., follows on the ingestion of mercury depends primarily on the condition of the mouth and teeth, the actual amount administered playing but a secondary part in its production.

Total number of patients treated for syphilis, 468. Twenty-three (4·91 per cent) were readmitted, having relapsed after treatment. Only eleven of these twenty-three cases had the full course of treatment; the remainder were admitted prior to the middle of February, that being the date when we were first supplied with "kharsivan."

In conclusion, I beg to acknowledge my indebtedness to Lieutenant-Colonel F. Ashe for his kindness in permitting me to publish the details of the above cases.

GUNSHOT WOUND, PERICARDIUM: PYO-PERICARDIUM WITH GAS INFECTION.

By Major W. J. P. ADYE-CURRAN.
Royal Army Medical Corps.

Pte. W., aged 22, was admitted to No. — General Hospital on October 9, 1916, with small round healed wound over ninth rib left side post-axillary line. There was no wound of exit.

Previous History.—Wounded September 27, 1916, with, he stated, a rifle bullet. He was detained at a Casualty Clearing Station for ten days and sent here. He coughed up blood after being wounded.

Condition on Admission.—The case was looked upon as being a simple penetrating wound of the chest, and except for some crepitations at the left base, nothing further was noted. October 14, 1916: A very loud exocardial sound was heard over the pericardial area of a sharp tapping nature. This was synchronous with the heart-beat and regular, and obliterated the heart sounds except over pulmonary region. The pulse was noticed also to be quick and irregular. The area of cardiac dullness appeared normal and was marked out with pencil on the chest wall. The patient's facial aspect was anxious. There was a cachectic tinge of the skin. October 15, 1916: The area of cardiac dullness had completely disappeared as also the abnormal sound. The heart sounds were inaudible. Sir J. Rose Bradford, K.C.M.G., C.B., kindly saw the case and expressed the opinion that the loss of cardiac dullness indicated a gas infection of the pericardium and advised an exploratory puncture. An X-ray photo showed in the antero-posterior plane a piece of metal, which had the appearance of a piece of shell the size of a hazel-nut towards the base of the heart. This could not be identified in the lateral view. The pericardium was also shown much distended and extending well into the right chest.

Operation the same evening after injecting two per cent novocain and adrenalin; a spinal trochar and cannula was introduced through the fifth intercostal space an inch from the sternum into the pericardium. On withdrawing the cannula there was an escape of gas which could be distinctly heard, and which had a very foul smell. It was then decided to drain the pericardium. The patient's condition did not justify a general anæsthetic. Under local anæsthetic, novocain and adrenalin, after blocking the intercostals, a flap was turned upwards off the chest and the sixth costal cartilage excised. The pericardium, which was greatly thickened, was opened and about eight to ten ounces of foul smelling yellow pus evacuated. A search was then made with a finger for the foreign body, but it could not be found. A quantity of defibrinated blood-clot was found lying in the upper right part of pericardium. A drainage tube was sewn to skin, the end lying just inside the opening of the pericardium. The patient felt nothing of the operation up to the opening of the pericardium, which caused an uncomfortable but not acute pain in the chest. An opening only about two inches long was made for this reason. Profuse discharge of pus continued from the pericardium, which was irrigated with eusol at blood-heat twice daily. Pulse very variable. Much improved after irrigation. October 28, 1916: The pericardial opening had contracted, preventing free drainage. Under a general

anæsthetic, open ether, the pericardial wound was enlarged to about three inches. The surface of heart had lost its shiny appearance and was adherent to pericardium near the apex. Another search was made for the foreign body without result. For four weeks a profuse suppuration continued. At first it contained a Gram-positive bacillus and cocci, and later streptococci. The pericardium was still irrigated twice daily, the fluid being ejected forcibly by the heart-beat. The patient was kept raised and leaning toward the left side, though he could not stand this position for long periods. The urine was found to contain albumin, blood-cells and casts, and there was slight œdema of the legs and ankles. The patient's appetite was extraordinarily good, and he was most hopeful and interested in his own case. October 28: Had a rigor, temperature 103·2° F. Sweated profusely and generally appeared to be suffering pain. Considerable toxæmia, though there was nothing in his condition to account for this except the continued suppuration which was markedly lessening daily. November 4: Another rigor and condition appeared one of septicæmia. November 5: 100 cubic centimetres of eusol given by vein, rigor followed. November 7: Delirious at times and appeared very ill. November 8: Eusol 100 cubic centimetres intravenously, temperature lower. November 9: Signs of fluid left pleura, specimen quite clear and sterile. Very persistent hiccough. Tinct. iodine minimum doses repeatedly. November 10: Hiccough severe. November 11: Severe diarrhœa, condition grave. Starch enema and catechu mixture. November 12: Delirium severe, diarrhœa. November 13: Severe diarrhœa, pulse 140. November 15: Ten ounces pleural fluid aspirated, left pleura, turbid. Fall in temperature. Case handed over to No. 7 Canadian General Hospital.

Officer Commanding No. 7 Canadian General Hospital kindly sent me the following note of the progress of the case:—

November 21, 1916: The fluid had accumulated again in the left chest. Patient's general condition improved. November 29: Chest aspirated, seventeen ounces of turbid fluid withdrawn. December 3: Pathologist reported streptococci in pleural fluid. The albumin and casts had disappeared from the urine. Œdema of legs and flanks markedly decreased. Discharge from wound lessened. December 7: Both upper arms swollen and tender (phlebitis). December 23: Swelling of arms lessened. Breath sounds audible over left base. January 1, 1917: General improvement. Slight discharge from wound which was contracted to a sinus-like tract. Some irregularity in cardiac sounds with a wide area of cardiac impulse. Temperature below 99° F.; pulse 104. February 1: Patient sitting up in chair for three hours daily. February 8: Transferred to England. Condition good. Urine secretions normal. Slight œdema of ankles. Some dullness over left base with fine crepitations. Phlebitis of arms recovered. Temperature 99·6° F.; pulse 116; respiration 26.

Note.— Since he has returned to England I have had several letters from him saying he was improving, but that there still was discharge from the wound.

THE SCABIES PROBLEM ON ACTIVE SERVICE.

By Major H. MacCORMAC, M.D., F.R.C.P.
Royal Army Medical Corps.
AND
Captain W. D. D. SMALL, M.D., F.R.C.P.E.
Royal Army Medical Corps.

Every man incapacitated from duty—it matters not by what means—is a gain to the enemy. From the point of view of the Army and of military efficiency, those diseases ought to rank highest in importance which cause most "casualties." Judged from this aspect, diseases of the skin occupy one of the premier positions, for under the conditions of trench warfare it is inevitable that they are met with in large numbers. The exact extent of the wastage for which they are responsible is known only to the authorities, but it is apparent to all that—since many are contagious—successful efforts directed towards prevention and cure will amply repay by a very large gain of fighting men.

Of the skin diseases found amongst soldiers—excluding pediculosis—scabies is by far the commonest. When uncomplicated, its effects incapacitate a man but little; but in consequence of the hard conditions of active service secondary pyodermic complications are frequent and severe, and these are a very real cause of prolonged sickness. In contrast to the three or four days required for the complete cure of a simple case, we have found that when pyodermic complications are established the period necessary is greatly prolonged—the average stay in hospital over a large number of cases being 31·67 days. It should be explained that this figure does not include either treatment elsewhere—which sometimes amounted to many weeks—or the days spent in journeying to the base.

Scabies as seen amongst soldiers differs in type from that usually met with in civil practice, and unless the differences are appreciated many cases will not be recognized, leading to dissemination by undetected "carriers." The eruption is distributed much in the usual fashion, the sites of election being the wrists, hands, penis, lower abdomen, anterior axillary borders, elbows, and buttocks. The characteristic interdigital burrows, however, are but rarely seen: they were present typically in only thirteen[1] of our cases. Vesicles are much more common, though in quite a number of cases the hands are entirely free from lesions of any sort. A correct diagnosis can only be made after inspection of the whole body. In the majority of cases the penis is involved, and the discovery of papules or crusts on either skin or mucous membrane is almost pathognomonic. This serves in doubtful cases to distinguish scabies from the acute pediculosis so frequently seen, and is for this reason an extremely useful diagnostic symptom.

Besides the primary lesions of scabies, others due to secondary coccal infection are common and important. They may be so severe as to mask the primary disease and render it liable to be overlooked. Most common is a variety of impetigo distributed in a characteristic manner on the lower buttocks, elbows,

[1] This figure does not include cases where one or two indefinite lesions could be found after prolonged search: including these, the percentage would be about 45. It is our impression that lately burrows have been observed with more frequency than some months ago.

and knees. The presence of pustules or crusting on one or all of these regions should at once suggest scabies and lead to search for the more primary lesions elsewhere. Occasionally the impetigo becomes very widely spread, but even in such cases a correct diagnosis can be made by observing the characteristic grouping on the regions indicated. In numerous cases the impetigo lesion extends more deeply into the dermis and takes the form of an ecthyma. Occasionally a linear variety of impetigo is present.

Boils and other follicular infections of the skin are frequent, and tend to recur indefinitely so long as the primary scabies remains uncured. No case should be considered as one of simple furunculosis until itch has been carefully sought for and excluded—or, if found, until the patient has had a thorough course of sulphur treatment.

Some degree of adenitis is the rule in all cases of scabies which have developed any form of coccal infection. The glands of the groin are those most frequently involved, and suppuration in this situation forms a not uncommon and often troublesome complication. In the upper limb severe lymphangitis with foci of suppuration seems to constitute the corresponding lesion. These septic foci usually occur in the upper third of the limb, and on its inner aspect.

Dermatitis as a complication of scabies is most frequently the result of unsuitable, misapplied, or too long continued treatment. In these instances, the abdomen, thighs and arms are the commonest sites—and all degrees from simple erythema to moist weeping or exfoliating eczema may be observed.

Subjects of the seborrhœic diathesis seem particularly liable to this complication.

Localized dermatitis may originate in a group of vesicles or other scabetic lesions, which, together with the intervening skin, have become irritated by scratching: considerable local thickening of the skin becomes superadded and the result is an area of chronic infiltrative dermatitis—in part scabetic and partly artefact. This is most commonly seen in the flexure behind the knee or on the outer aspect of the thigh.

In its diagnosis, scabies must not only be distinguished from the pyodermic conditions apt to be associated with it, but also from pediculosis, pompholyx, syphilis, and an unusual papular urticaria.

From pediculosis, the diagnosis is chiefly made by the difference in distribution, and the absence of infection of hands, wrists and penis. Not only may the two conditions be associated, but the papule,[1] common to both, can only be distinguished with extreme difficulty.

The vesicular lesions of pompholyx are much more deeply seated, more numerous, and more difficult to rupture than those of scabies, and are chiefly limited to the hands and feet.

A single large scabetic lesion on the penis may give rise to question as to whether or not the patient is suffering from venereal disease, and the eruption on the body may be mistaken for a secondary rash; the diagnosis is not always so easy as might be supposed, particularly if risks of venereal infection have recently been incurred. Here also the two conditions may occur together.

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[1] Pediculosis as usually seen in France occurs in an acute form and characteristically presents a papular type of eruption.

A type of papular urticaria which we have not observed in civilian practice may simulate scabies very closely. In this condition, small red papules occur over the body generally but particularly on the abdomen and anterior axillary folds; wheals, or some degree of dermographia, may often be found or induced. Again, the wrists, hands or penis are not involved, and the disease tends to disappear suddenly, while relapse is frequent.

The methods of combating scabies fall under the two headings—prevention and cure.

(*a*) *Preventative Measures.*—Scabies it is generally accepted is rarely contracted excepting after prolonged and intimate contact with infected material, for the acarus does not wander afield like the louse. This being so it is difficult at first sight to explain how the soldier gets the disease. His opportunities for removing clothing and coming into contact with an infected medium are few and are only fully possible when in rest. All the evidence—particularly the occurrence of epidemics amongst officers sleeping in the same dugout, or amongst groups of men quartered together—points to blankets as the chief means of disseminating infection. Prevention then should include frequent disinfection of blankets by such a method as the Clayton sulphur vapour apparatus, or some other accepted form of sterilization. A few cases seem to be contracted from horses, and the protection of the soldier therefore includes efficient treatment of the horse. Great stress is laid by French writers [1] upon the venereal origin of scabies, and this possible source of infection requires to be kept in mind.

Of the greatest importance is the early detection of every case of scabies. Each case undetected acts as a "carrier," and is the potential starting point of an epidemic. Frequent and thorough inspection of the individual soldier is the only means whereby early diagnosis can be assured. Infected men should be segregated and treated as speedily as possible, for—in addition to preventing dissemination—early diagnosis enables treatment to be instituted while the disease is still uncomplicated, and renders its course short—correspondingly conserving man-power.

(*b*) *Curative measures* should include exposure of the parasite and its ova and their destruction by remedies short of producing dermatitis. The subsequent disinfection of clothing and bedding to prevent reinfection must never be omitted.

Sulphur, it is generally acknowledged, is the most efficient remedy for the treatment of scabies—and certainly by reason of its procurability and cheapness it is that most suited for the extensive requirements of an army. It is a remedy, however, which requires careful and intelligent application, and care must be taken that the patient receives neither too little nor too much treatment—the former leaving him uncured, the latter producing dermatitis, and so increasing his period of unfitness. How this remedy should be employed is already so well known that a repetition of the details would seem unnecessary had it not been for the recent reintroduction of the sulphur vapour method. This form of treatment so scathingly condemned by Hebra [2] in the first half of the last century, seems to have again obtained a certain vogue, in spite of the fact that it was so completely discredited nearly a hundred years ago. Indeed, it has so long been regarded as one of the barbarities of the Dark Ages that it is rarely mentioned in modern text-books. We have had opportunity of observing a

number of cases treated by this method. Some have suffered from severe dermatitis, numerous others have been uncured. The history given by some of these latter is instructive. After the vapour treatment, the itching was relieved, and improvement appeared to take place in the eruption. The patient seemed cured, but after a period of about two weeks the disease again became active, so that it would appear that in these a false sense of security had been obtained and a "carrier" discharged to further disseminate the infection. That a certain number of cases may be cured by this method is probably correct, but its uncertainty and the special danger of dermatitis from which it is inseparable give us no option but to emphatically condemn its use.

Liquid preparations of sulphur, such as Vlemingx's solution (liquor calcis sulphurata) are undoubtedly efficient agents for the cure of scabies, but they also are particularly liable to cause dermatitis, and considerable experience is necessary for their satisfactory employment.

For its simplicity and certainty, no method is so suited for general use as that of simple inunction with sulphur ointment (B.P.), and admirable results will invariably be obtained, if the treatment is conscientiously carried out and with due regard to the necessary details. It must, however, be insisted that the method of application is of paramount importance. This may be briefly outlined as follows:—

(1) On the first day of treatment, the patient is given a hot bath, and provided with plenty of soft soap, and a large moderately stiff nail-brush.

(*a*) Before entering the bath, he rubs himself thoroughly all over with the soap, massaging it into the skin, and paying particular attention to the fingers, toes, wrists, penis and axillæ. This should be continued for at least ten minutes.

(*b*) He then enters the bath, which should be both long enough and contain a sufficiency of water to permit of immersion to the neck. After first steeping for fifteen minutes, he scrubs himself vigorously all over with the nail-brush for the purpose of opening burrows and vesicles. In cases where the eruption is particularly severe or painful, this part of the procedure may be correspondingly modified. After the bath, a final inspection should be made, when any unruptured vesicles can be opened with a surgical needle.

The bath is given with the sole object of opening up the haunts of the acarus and exposing it to the action of the parasiticide to follow. It is obvious that shower or steam baths cannot fulfil this purpose, and examination of a patient so treated will reveal the burrows and vesicles still intact.

(2) After removing the soap and drying, a liberal quantity of sulphur ointment (B.P.) is provided, with which the patient rubs himself vigorously all over, from the neck downwards, special attention being paid to the affected parts and to the fingers, wrists, genitals, axillæ, toes and ankles. The ointment must be liberally applied and thoroughly rubbed in, so that when finished he should be literally "soaking" in it.

The inunction is to be repeated in this manner twice daily for three days—i.e., until the patient has had in all six applications. Each must be complete, and for this it is obvious that all clothing be first removed. The treatment of limited parts of the body is useless.

(3) Finally, on the fourth day, but not before, a second bath is given, and all

the patient's clothing and bedding sterilized to prevent reinfection. Even such articles as wrist straps, strings of identity disks, gloves, etc., should be included.

The vast majority of cases, unless there be secondary complications, will be found cured after three such days of treatment. Should any doubt exist, 2 per cent beta-naphthol in vaseline may be used daily for four more days. Only under exceptional circumstances should sulphur ointment be applied for more than three days, since its continued use is liable to cause severe dermatitis. Indeed, in susceptible persons, a mild degree may be occasioned by the three-day treatment. This is usually easily cured by zinc ointment or Lassar's paste. At the completion of treatment, the existence of some degree of itching is not uncommon; this is due to the remedy and does not indicate failure of cure; it will rapidly pass off, and should be disregarded. Some days also will probably elapse before all the lesions of scabies have disappeared, although the man is no longer infective. A certain degree of pigmentation may persist indefinitely.

Conclusions.

(1) Scabies in soldiers differs in type from the ordinary form of civil practice, particularly in the frequent absence of burrows or other lesions upon the hands, and in the great liability to pyodermic complications.

(2) The primary disease is often masked by complications. In every case of impetigo, boils, dermatitis or inguinal adenitis, scabies must be carefully excluded before the diagnosis is considered complete.

(3) Cases of scabies complicated by any of the above necessitate about ten times as long a period in hospital as do simple cases. Therefore early diagnosis and treatment, before secondary diseases have become established, are of the greatest importance.

(4) Regular medical inspection is essential for the detection of "carriers." Infected men should be segregated and treated at the earliest opportunity. Horses may also require inspection. Blankets, being the chief source of spread, should be sterilized as often as possible.

(5) Treatment by means of sulphur vapour is a method harmful often to the patient, and at all times dangerous to the community in that it manufactures a class of scabies "carrier." In the interests of the Army it should be discontinued.

(6) The method of treatment most suitable to service conditions on account of efficiency, simplicity, and cheapness, is inunction with sulphur ointment, but this must be carried out in a methodical and thorough manner, and with careful attention to the necessary details.

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[1] Milian. *Paris Médical*, May 6, 1916. p. 426.
[2] Hebra on "Diseases of the Skin" (New Sydenham Society), ii, p. 241.

ON THE TRANSMISSION OF THE SUBTERTIAN MALARIA PARASITE (*PLASMODIUM FALCIPARUM*, WELCH, 1897) BY EGYPTIAN ANOPHELES.

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Having been unable in the past literature of malaria to find any records of the actual development of the subtertian parasite in the local species of Anopheles found in Egypt, I have thought it advisable, on account of the prevalence of this form of malaria in certain parts of Egypt, as well as of the prevention of its spread amongst the troops stationed there, to place a short research I carried out in the autumn of 1916 on record.

Five well-recognized species of Anopheles have been recorded (Gough, *Bull. Ent. Res.*) as occurring in Egypt; these are:—

(1) *Anopheles* (*Cellia*) *pharoensis* (Theobald).
(2) *Anopheles squamosus* (Theobald).
(3) *Anopheles* (*Myzomyia*) *turkhudi* (Liston).
(4) *Anopheles* (*Pyretophorus*) *palestinensis* (Theobald).
(5) *Anopheles* (*Myzorhynchus*) *mauritianus* (Grandpré).

Of these for our present purpose *squamosus* can be left out of count, as it has been recorded but once—by Theobald. The remaining four species, however, appear to be widely distributed in Egypt. On the Canal zone I have found *pharoensis*, *turkhudi* and *mauritianus*. In the Fayoum and Western Oases, *pharoensis*, *turkhudi* and *palestinensis*. In the Cairo district, *pharoensis* and *turkhudi*. With the exception of *turkhudi*, few direct transmission experiments with the subtertian parasite appear to have been performed with any of these species in India or any other country in which they occur.

I will now give a short account of previous evidence with reference to the spread of malaria by these species.

(1) *Anopheles* (*Cellia*) *pharoensis*.—The transmission of the benign tertian parasite (*Plasmodium vivax*, Grassi and Feletti, 1890) has been followed out in this species by Newstead, Dutton and Todd, but no work in this direction appears to have been done on the subtertian parasite (*P. falciparum*) in this species.

(2) *Anopheles* (*Myzomyia*) *turkhudi*.—This species comprises the following, all of which are synonymous, or are at the most geographical variations:—

Anopheles multicolor, Camboulin, 1902.
Anopheles impunctus, Dönitz, Egypt, 1902.
Pyretophorus chaudoyei, Billet, Algeria, 1903.
Pyretophorus myzofacies, Theobald, India, 1907.
Pyretophorus nigrifasciatus, Theobald, Algeria, 1907.
Myzomyia azriki, Patton, Aden, 1905.
Myzomyia hispaniola, Theobald, North Africa and Spain, 1903.

Zygotes of the subtertian parasite have been found by Stephens, Christophers, and James in the stomach of this insect in India, and it has been regarded as one of the carriers of this parasite in that country (James, *Sci. Mem. Gov. India*, 1902, No. 2, p. 79).

(3) *Anopheles* (*Pyretophorus*) *palestinensis*, synonymous with *Pyretophorus sergenti*, Theobald, Algeria.—Under the latter name Ed. and Et. Sergent have

found sporozoites in this species and regard it as a common carrier in Algeria and Spain. It appears to be distributed in Algeria, Spain, Cyprus and Palestine, as well as in Egypt, where, however, its range is confined to the Western Oases—Kharga, Dahkla, Baharia and Siwa Oases. Though its distribution intimately corresponds with that of subtertian malaria in those localities no direct evidence against it ever appears to have been recorded in Egypt. This species is very closely allied to *turkhudi*, but differs in having the terminal segment of the palp tipped with white in place of black.

I found this species in January, 1917, breeding abundantly in the warm wells in the Dahkla Oasis (the water temperature varying from 80° F. to 120° F.). Adult insects were then abundant in camp, and although the temperature was as low as 1° C. (34° F.) they appeared to be capable of conveying the subtertian parasite to freshly arrived troops, a proportion of whom contracted the disease ten days after arriving in the Oasis. As it is generally stated (Hindle, p. 162) that the development of this parasite ceases below 18° C. (64° F.) the epidemiological evidence I have just recorded requires further elucidation.

(4) *Anopheles* (*Myzorhynchus*) *mauritianus* is a rare mosquito in the interior of Egypt and appears to breed according to my experience in brackish water. Owing to its comparative rarity in malarial districts it is doubtful whether it plays any part in the actual transmission of malaria in Egypt, and it is regarded by Ross, as it occurs in Mauritius, in this light.

During the months of October, November and December, 1916, an opportunity presented itself in Cairo of carrying out the transmission of the subtertian parasite in species of Anopheles which were, owing to the pools formed by the overflowing of the Nile, most abundant at that time, namely: *Anopheles* (*Cellia*) *pharoensis; Anopheles* (*Pyretophorus*) *turkhudi.*

Large numbers of freshly-hatched insects were caught and subsequently fed on a Bulgarian patient from Salonika, whose blood, although he was not suffering from fever, contained large numbers of crescents. These were estimated by means of a Thoma-Zeiss counting apparatus at about 600 per cubic millimetre of blood. The exflagellation of these crescents readily took place from ten to fifteen minutes after blood had been drawn.

Owing to the active co-operation of the patient it was found possible to feed the infected Anopheles daily for twenty-two days from the commencement of the experiment on his blood, and a certain number of insects were dissected daily from the second day after feeding onwards. The insects were fed in gauze cages, and were subsequently placed in a damp atmosphere in a chamber kept constantly at a heat of about 25° C.

In order to keep the atmosphere sufficiently damp—and I wish to make clear it is a very essential point for the life of the mosquito—the cages were covered every night with damped Chardin filter paper. Out of the large number successfully fed thirty-six specimens of *Anopheles* (*Cellia*) *pharoensis* survived after the fourth day, and their stomachs were dissected and stained. In one specimen only out of these thirty-six were any developmental forms found. In this two large oöcysts (measuring forty-five microns by thirty microns) containing sporozoites were found on the fifteenth day after the original feed of crescent-containing blood.

It would appear therefore that this species can occasionally act as a trans-

mitter of the subtertian parasite, but probably under natural conditions it is not a significant factor in this respect.

On the other hand only three specimens of *Anopheles* (*Myzomyia*) *turkhudi* survived after the third day, and only one to the tenth day after its first feed. On dissection the latter specimen showed an extensive development of pigmented oöcysts in the stomach, no less than ninety-six being counted with a $\frac{1}{6}$ inch lens in a permanent specimen stained with Giemsa. They measured on an average twenty-four microns in diameter.

I have submitted specimens of Anopheles of each batch I experimented with to Mr. G. Storey, B.A., F.E.S., of the Entomological Section, Ministry of Agriculture, Egypt, and I am indebted to him for the confirmatory identification of the species.

Conclusions on Experimental Grounds.

(1) *Anopheles* (*Myzomyia*) *turkhudi* is an efficient definitive host of the subtertian malaria parasite in Egypt.

(2) *Anopheles* (*Cellia*) *pharoensis* can act as an inefficient and occasional definitive host for the subtertian parasite. This fact is of interest in view of the prevalence of its congener *Anopheles* (*Cellia*) *pulcherrima* in Mesopotamia where it is regarded as a probable carrier.

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THE SUCCESSFUL CONSERVATIVE TREATMENT OF EARLY GAS GANGRENE IN LIMBS BY THE RESECTION OF INFECTED MUSCLES.

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Among the points brought forward by Colonel Cuthbert Wallace in an article on "Gas Gangrene,"[1] some months ago were the following:—

(1) It is rare to meet gas gangrene without muscle injury.

(2) It is chiefly a disease of muscles, and is rarely dangerous unless muscle is involved.

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[1] "Gas Gangrene as seen at the Clearing Stations," *British Medical Journal*, September 16, 1916.

(3) The lesion in its early stages may be described as a longitudinal one, running up and down the wounded muscles from the seat of the lesion. Muscles and groups of muscles are involved while others escape.

(4) It is rare to find all the muscles of a segment of a limb involved, save in a segment distal to one in which the main blood supply has been cut off. Thus the whole leg dies and becomes gaseous when the femoral artery has been blocked in the thigh.

(5) There is little tendency for the infection to pass from one muscle to another. This is well shown in amputation stumps where one muscle dies and becomes gaseous, while the rest of the cut muscles remain healthy.

From our own observations in clearing stations over a period ranging up to twenty-one months, it is abundantly clear that these points cannot be controverted. In no case have we seen gas gangrene commencing as a subcutaneous infection; injured muscle is in all cases the initial focus—the appearance of crackling in the subcutaneous tissues being a secondary phenomenon due to extravasation of gas from the infected muscles below.

In view of this it has been our aim to model our treatment on the following lines: to explore the primary focus with a view to attempting to arrest the infection in the muscle or group of muscles involved by *resection* of the infected areas. Such resection may, as will be seen from the cases, involve a part or the whole of single muscles or groups of muscles.

Resection should be limited to cases in which the main vessel of the limb is intact and should be replaced by amputation where the operation must be so extensive as to be likely to give a limb which would be of less value than an artificial limb. Extensive resections however may be performed regardless of the ultimate utility of the limb, as a life-saving operation, it having been quite clear in some of the cases recorded below that an amputation as in Case 13, or a further amputation in Cases 7 and 9 could not have been carried out without very grave risk to life.

Resection should extend until muscle is reached, which has the following characteristics: (1) The colour is unchanged; (2) the contractility is normal; (3) a good blood supply is present, as indicated by free bleeding from the cut surface. Experience has shown that even if such muscles are, as they may be, slightly infected, free drainage and an open wound will arrest further development of the condition.

The treatment of cases after resection is carried out on the following lines: (1) The dressings are reduced to the absolute minimum, i.e., one or two layers of gauze only being placed over the wound so as to allow free access of air and if possible sunshine to the wound region; (2) constant or intermittent irrigation of the wound by some modification of the Carrel method—eusol, saline or peroxide of hydrogen being used as the irrigating fluid.

In connexion with the cases described below, the point must be specially emphasized that gas gangrene had already developed in some of the patients, though they were admitted a very short time after being wounded. In fact in units at the front established gas gangrene has to be treated *ab initio* in addition to gas gangrene developing at a later stage.

We are indebted to Colonel Cuthbert Wallace, consulting surgeon to this Army, for advice and assistance in the treatment of these cases, and to Captain

J. W. McNee and Lieutenant J. S. Dunn, of No. 3 Mobile Laboratory, Royal Army Medical Corps, for the bacteriological and pathological investigations they have made in the cases.

Case 1.—Lieutenant F., wounded 4 p.m., on November 2, 1916, by rifle grenade fragment; admitted at 7.15 p.m., the same day, i.e., three and a quarter hours after being wounded. When admitted he was clearly very ill, although he arrived as a walking case; his pulse was 120, of poor quality and irregular, his tongue was furred and his features pinched and worn. There was an irregular wound the size of a threepenny-piece on the posterior aspect of the right arm, just above the internal condyle of the humerus—there was no exit. The whole upper arm appeared swollen; it was crepitant to the touch and gave a tympanitic note on percussion. The crackling was most marked on the inner aspect of the arm and extended as high as the anterior axillary fold above in front but to not quite so high a level behind. He was given a general anæsthetic at 8 p.m. The wound of entry was first excised, much damage to the triceps was found locally and the projectile could be felt in the inner side of the vessels in the front of the arm at the junction of the lower and middle thirds; a second incision was made over this when the subcutaneous tissues were found to be œdematous and the biceps muscle showed evidence of gas infection. A further incision was accordingly made along the whole length of the biceps muscle from its origin to attachment; this showed the following conditions: some bruising of the inner border of the muscle at the situation of the projectile; discoloration of the inner half of the muscle varying from a dull red colour at either end to a deep plum colour in the centre. Over the discoloured area bubbles of gas were present under the fascia covering the muscle. This half of the muscle did not react to tap stimulation, whereas the outer half was normal in colour and contracted sharply on stimulation. As this appeared to be an infection localized to the inner half of the muscle, the latter portion was resected in its entirety. A tube drain was inserted into the posterior wound; the anterior wounds were left open, small tubes being inserted for continuous eusol irrigation, which was commenced immediately after the operation was completed. The pulse dropped to 88 six hours afterwards, and never rose above this again; the wounds remained clean without pus formation, and on November 9, 1916, were closed by secondary suture, and the eusol irrigation was discontinued. The wounds remained healthy and were practically healed when he was evacuated to the base on November 17, 1916. He had already very fair power in his arm, and the remaining portion of the biceps could be felt contracting.

Pathological Report.

An anaerobic gas-forming organism was grown from the excised muscle, which on histological examination showed hyaline degeneration of groups of fibres with separation. This condition represents a very early stage in the process of gangrene. Long bacilli could be recognized between the muscle fibres, but were very scanty.

Note.—The extreme rapidity of the infection in this case is remarkable, occurring as it did within three and a quarter hours of the time of injury, in fact it is the earliest case that we have ever seen. There is no doubt that if any delay had occurred in the treatment the consequences would have been disastrous owing to the virulence of the infection, and the patient would have lost his limb if not

his life. The treatment by resection of half of the muscle rather than the whole, which at first sight would seem to have been the better course, was due to the fact that the infection is known to spread longitudinally along the length of the fibres, and that transverse spread is a late phenomenon occurring in the more advanced stages. The differentiation between living and dead portions of the muscle was made from the presence or absence of contractility, it being well established that heavily infected muscle is non-contractile. (C. H. S. F.)

Case 2.—Lance-Cpl. D., wounded by shell fragment on July 2, 1916, admitted twenty-four hours later, when the following condition was found. Temperature 102·6° F., pulse 118; there was a through-and-through wound in the middle of the left upper arm; the arm was greatly swollen in the neighbourhood and tender on palpation. Subcutaneous crepitation was present. At the operation, which was performed at once, the biceps muscle at the site of the wound was found to be in a state of "black death," and was full of gas; for two inches above and below this the muscle was not contractile, and in the "red death" stage. A long incision was made over the whole length of the biceps and the entire muscular portion was excised. The wound was dressed with gauze soaked in peroxide until all oozing had ceased and then was left exposed to the air and sun under a single layer of gauze. The patient vomited a good deal for two days after the operation, but on the third day this ceased and his temperature and pulse dropped to normal and remained so. No other muscle was affected, and the resection was entirely curative and conserved his limb. He was sent to the base on July 11, 1916, with a healthy granulating wound, and a satisfactory report on his condition was received from England on July 26, 1916. (G. E. N.)

Case 3.—Pte. O., admitted at 7.30 p.m., on October 29, 1916, suffering from multiple shell wounds received the same afternoon. His general condition was good: there was a penetrating wound of the right chest, an in-and-out wound of the right calf and a penetrating wound of the right biceps muscle. The next day he was given a spinal anæsthetic and the wound in the leg was incised and drained. The next day his condition was not so good and he complained of pain in the right arm; pulse 106, temperature 101·8° F. The wound in his arm was the size of a sixpence and was situated over the centre of the biceps muscle. The arm was swollen and the skin was tense and slightly discoloured in the region of the wound. On gentle pressure a small amount of dirty serum with gas bubbles in it escaped from the wound. A general anæsthetic was given and the biceps muscle exposed by a long incision. In the middle third of the muscle deeply situated there was a small cavity containing a fragment of shell; above and below this for a distance of two inches was an area of dead crepitant muscle. The whole necrotic area was excised until healthy muscle was reached which bled on section. The wound was left freely open and dressed with a eusol gauze. He made a straightforward recovery and was evacuated to the base on November 12, 1916. The wound in his arm was then healthy and ready for secondary suture.

(*Note.*—In this case immediate improvement followed resection of the infected muscle.—H. D.)

Case 4.—Pte. G., wounded July 24, 1916, admitted the same day. Shell wounds right buttock, right calf and skull. Immediate operation, trephining, wound of buttock and calf drained and piece of metal removed from soleus

muscle. Twenty-four hours later the calf was very swollen and tender and gas bubbled from the wound on pressure. Temperature 102° F., pulse 112. A second anæsthetic was given and the calf opened up from the lower part of the popliteal space to three inches from the ankle. The gastrocnemius was split down the mid-line and found to be healthy and contractile; the soleus beneath along the track of the projectile was found to be in the black death stage of gangrene, it was crepitant and no part of the muscle was contractile. As much as possible of the muscle was cut away and the wound was flushed out with peroxide and left open to the air, the limb being slung in a cradle for this purpose. The patient was given 100 cubic centimetres of five per cent eusol solution intravenously at the end of the operation. Rapid improvement followed; the temperature and pulse dropped to normal on the next day and he was evacuated to the base on July 30, 1916, with a normal temperature and pulse. The head wound had healed and the other wounds were clean. A note from the Base Hospital stated that he was sent to England on August 5, 1916, having made an uninterrupted recovery.—G. E. N.

Case 5.—Second-Lieutenant G., wounded in the left leg by a rifle bullet at close range at 2.30. a.m., on October 11, 1916, admitted at 9.0. a.m., and operated on immediately. There was a through-and-through wound in the upper and outer part of the left calf—the wound of entry and exit being of about the same size and just large enough to admit the tip of the finger. The wounds were excised and the wound freely drained by means of two large tubes after irrigation with eusol; the wound appeared to be a comparatively trivial one. Eighteen hours later his pulse which had been previously normal ran up to 120, and the patient showed signs of a profound toxæmia; the upper part of the leg was swollen and tender and the skin was glazed; there was no subcutaneous crackling. A further anæsthetic was given and the entire track of the wound, which was about three inches long, was laid open by division of skin and intervening muscles. The peroneus longus and brevis muscles were found to be considerably lacerated, they were altered in colour, crepitant and non-contractile. The dead portions were cut away until healthy bleeding muscle was reached which contracted on stimulation. The wound was left open and treated with continuous eusol drip irrigation. Immediate improvement followed, the pulse dropping to normal within a few hours. He was evacuated to the base nine days later with a healthy granulating wound. Anaerobic gas-forming organisms found in the dead muscle removed.

Note.—This case is of interest as the infection commenced in spite of free and early drainage. Resection of the infected area cut the process completely short.—C. H. S. F.

Case 6.—Cpl. E., was admitted to the station at 5.30 p.m., on August 27, 1916, having been wounded by a shell fragment at 7 p.m. the previous day. On admission he looked flushed, his tongue was dry and his pulse 120, temperature 103° F. There was an entry wound the size of a shilling over the anterior tibial group of muscles at the junction of the middle and lower thirds of the leg; the wound of exit was two inches above the ankle-joint on the posterior aspect of the limb in the middle line. The missile had passed between the two bones of the leg. The limb was very swollen in its lower half and the skin had a white tense glazed appearance; there was considerable pain on palpation. No

subcutaneous crackling was elicited. Under the anæsthetic on raising the limb gas bubbles escaped from the anterior wound with some foul stinking serum. The skin wound was excised and the anterior tibial group of muscles were explored by a six-inch incision. The extensor longus digitorum muscle was exposed—this had been divided across by the missile; the retracted ends showed an opaque sheath with underlying dead non-contractile muscle for an inch on either side of the wound. The dead portions were cut away until normal vascular muscle was reached at either end. About two and a half inches in all were cut away from either end; the anterior tibial artery was exposed at the bottom of the wound but was uninjured. The extensor longus hallucis looked healthy and contracted well. The posterior muscles were not markedly damaged and were not interfered with. The wound was drained and drip irrigation with eusol commenced. The patient made an uninterrupted recovery and was evacuated to the base on September 3, 1816; his pulse was then 90 and his temperature 99° F.; the wounds were granulating.

An anaerobic gas-forming organism was isolated from the removed muscle which on section showed necrosis and irregular fragmentation of muscle fibres; there was œdematous thickening and leucocytic infiltration of the interstitial connective tissue; large bacilli were present in considerable numbers between the dead fibres.

Note.—This case clearly shows the longitudinal spread of the infection in the muscles and how free resection of the diseased muscle arrests any further spread of the infection.—H. D.

Case 7.—Pte. C. was wounded at 4 p.m., on July 29, 1916, by a shell fragment in the region of the left knee. On admission five hours later his condition was very bad owing to loss of blood, pulse 150. Six hours later after warmth and stimulation he was fit for operation and the wound was explored. There were two lacerated wounds on each side of the popliteal space which had evidently been traversed from within outwards; examination showed an extensive fracture of the head of the tibia involving the knee-joint. The pulsations of the main vessel could not be felt; gastrocnemius and soleus muscles were extensively lacerated. The wound was freely drained after irrigation with eusol. The next day his condition was satisfactory until 8 p.m., when his pulse was 120 and his temperature 102·4° F. The region of the wound was more swollen and the skin over the calf had the appearance of a bruise which was fading; percussion gave a tympanitic note over this area; there was no subcutaneous crackling. The knee-joint was also resonant from the presence of gas. The lower third of the leg was cold and there were several light purple patches over the dorsum of the foot. The leg was removed by disarticulation at the knee-joint; examination of the limb showed occlusion of the popliteal artery from a bruise wound and a large hole in the popliteal vein, the gastrocnemius and soleus muscles showed marked gas gangrene. He was much improved the next day, but on the following day his pulse was 120, and his temperature 101·4° F.; the lower third of the thigh was swollen, tender to the touch and resonant to percussion; it was evident that the infection was spreading up the muscles of his thigh. Under chloroform an incision was made up the back of the thigh and the semi-membranosus muscle was found to be gaseous and stinking. It was followed up to the middle of the thigh and resected at this point as the fibres were found to be healthy there.

The anterior aspect of the thigh was then explored and the sartorius muscle was found to be similarly infected in its lower part; the distal four inches were resected through healthy muscle. The wounds were left exposed to the air, being covered by one layer of gauze only; the wound was kept irrigated by a constant eusol drip. The thigh was slung so as to take the weight off the muscles of the back of the limb; suspension was effected by means of a gauze band attached to the exposed condyles of the femur. From this time on he commenced to improve, on the following day his pulse was 96 and temperature 101° F. On August 3, the stump of the popliteal artery, which was lying in a bed of sloughing muscle, commenced to ooze: in order to avoid a secondary hæmorrhage the femoral artery was ligated in Hunter's canal under local anæsthesia. After this he continued to improve and was evacuated to the base on August 15. The muscles were infected with a pure culture of *Bacillus perfringens*.

Note.—It was impossible even to attempt to save the limb in this case by resection of muscle in the first instance owing to the occlusion of the popliteal artery. When the secondary spread of infection occurred into the thigh muscles after the amputation his general condition became rapidly so bad that a further amputation would have been inevitably fatal. Resection of the infected muscle was the only satisfactory course to adopt, as proved to be the case.—H. D.

Case 8.—Pte. P., wounded August 28, 1916, admitted the same day. Through-and-through wound of left thigh with compound fracture of the femur in the upper third. He had had much hemorrhage before admission and was very collapsed on admission—pulse 140, temperature subnormal. Twelve hours later he had improved sufficiently for operation, both wounds were freely opened up, lacerated muscle, fragments of bone and the case of a bullet being removed. The outer wound was kept open with a silver retractor and both were lightly packed with eusol gauze. The limb was immobilized on a Wallace-Maybury splint and the wounds were syringed every two hours with eusol. Patient had a good night but next morning his temperature and pulse began to rise, he complained of pain in the limb and of great thirst. His tongue became dry and brown. On examination the limb was found to be swollen and tense and had a characteristic odour. There was no skin crepitation and no gas-bubbles from the wound. An immediate "chaff cutter" amputation just below the trochanters was performed and the adductor muscles were found to be in the red stage of gas gangrene, so they were completely excised from the stump. The wound was dressed for twenty-four hours in gauze soaked in peroxide and then left exposed to the air under one layer of gauze. Within two days the temperature dropped to normal and the pulse to 90; none of the other muscles in the stump showed any sign of being infected and he was evacuated to the base on September 15, 1916, with a granulating wound. A laboratory report confirmed the diagnosis of gas gangrene. This method of removing a group of infected muscle was also carried out in another case five days after amputation, the adductors here being also removed—no spread occurred in the other muscles.—G. E. N.

Case 9.—Lieutenant B., wounded at 1 a.m. on September 13, 1916, by shell, right leg shattered in lower third. Admitted at 3.30 a.m. the same day. Primary amputation by equal lateral flaps at the middle of the leg was performed at 4 a.m.; at the operation it was noticed that the anterior tibial artery was occluded, there was no change in the muscles which were contractile and

apparently healthy. The wound was freely irrigated with eusol and the flaps which were free were united loosely with three sutures. Free drainage was provided for by means of two tubes which also served for constant eusol irrigation which was started at once. The pulse before the operation was 90, at the end of the operation it was 88. Twenty-six hours later the pulse ran to 120, tongue became furred and dry and the patient was drowsy and obviously very ill. Examination of the wound showed mottling of the skin over the front of the leg, together with a tympanitic note on percussion over the same area; there was no crackling of the subcutaneous tissues. The wound was at once opened up, when it was found that the entire anterior tibial group of muscles were a brick red colour, non-contractile and quite dead. They were removed *en masse* and the wound was left open under constant eusol drip irrigation. The general condition remained bad for twenty-four hours with rapid pulse, drowsiness and persistent vomiting. Steady improvement then followed and the patient was sent to the Base on September 23, 1916, with a healthy granulating wound.

The removed muscles were found to be heavily infected with *B. perfringens* and other gas-forming organisms on culture.

Note.—The only other alternative in this case would have been a further amputation through the thigh, which it is doubtful if the patient would have survived; resection of the muscles avoided this additional danger and gave the patient a much more serviceable stump as the knee-joint was saved.—C. H. S. F.

Case 10.—Pte. P. was wounded by shell fragment on July 23, 1916; admitted July 24, 1916. On admission—temperature 103·6° F., pulse 120. There was a through and through wound of the left thigh, the entry being on the inner side three inches above the patella and the exit two inches higher up on the outer side. The exit wound was swollen, tense and tender, but did not crepitate. Both wounds were excised and united by incising the skin. There was some laceration of the edge of vastus internus; this was cut away, the muscle looked normal and was contractile. The rectus femoris was grooved on its under surface but appeared otherwise normal. The exposed inner edge of vastus externus was brick red and non-contractile and smelt strongly of gas gangrene. An area of four inches by two in the muscle showed signs of red death, the parts beyond were normal and contractile. A free skin incision was made and a piece of muscle eight inches by three enclosing the affected area was excised; the wound was dressed in gauze soaked in peroxide and later exposed to sun and air. The temperature and pulse steadily fell and by July 30, 1916, were 99° F. and 76 respectively. He was sent to the Base on July 30, 1916; the wound then was healthy and granulating and there was no sign of further gas infection. A report from the mobile laboratory stated that the infection was due to an anaerobic gas-forming organism.—G. E. N.

Case 11.—Pte. M., wounded on August 29, 1915, by shell fragment in the left groin: there had been much hæmorrhage at first, necessitating saline infusion in the Field Ambulance, where he was retained until mid-day on August 31, 1915. On admission his condition was grave: tongue brown and dry, pulse 130. There was a ragged wound of entry the size of a shilling, an inch and a half below Poupart's ligament and just to the inner side of the femoral vessels; the skin over the upper third of the thigh and for a hand's breadth above the wound was mottled and discoloured, and showed marked subcutaneous crackling. An

anæsthetic was immediately given and the wound opened up; it was found to extend upwards and outwards towards the anterior superior spine where a fragment of metal and khaki was found in the sartorius muscle, The muscle in this situation was dead and the upper third was cut away. The discoloured and crepitant areas were incised down to deep fascia and freely injected with hydrogen peroxide. No dressing was applied and the wound was kept constantly irrigated with hydrogen peroxide and left exposed to the air and sun. Rapid improvement took place and the man was evacuated to the base with a granulating wound twelve days later. No bacteriological examination was made, but there is no doubt about the condition.

Note.—At the time when this case came under treatment the significance of the muscle resection was not realized, but it seems at least probable that it cut short the process by removing the main focus of infection.—C. H. S. F.

Case 12.—Lance-Cpl. C., wounded in the left thigh by a shell fragment at 3.0 p.m. on March 13, 1916; admitted into hospital at 2.30 p.m. the following day. On admission he looked flushed: temperature 102° F., pulse 130. There was a jagged wound two inches in length on the outer aspect of the lower third of the left thigh. No exit wound. The lower part of the left thigh was swollen and tense and the overlying skin was shiny. There was tenderness on palpation and a tympanitic note was obtained on percussion; there was no subcutaneous crackling. Operation 5.30 p.m. the same day. A long incision down the outer aspect of the thigh exposed the vastus externus muscle, of which the lower half was markedly affected with gas gangrene. A portion of the muscle near the wound of entry was black in colour and the muscle fibres were diffluent. The lower part of the muscle was resected up to the middle of the thigh where healthy contractile muscle was found. As the muscle planes were separated by gas up to the level of the great trochanter the skin incision was carried up to this level. The whole wound was left wide open and dressed with eusol gauze. At 9.0 p.m. the same evening he was given an intravenous injection of eusol 100 cubic centimetres. His pulse dropped the next day and he showed a great improvement; this was maintained and he was evacuated to the Base six days later. Information was received from England a week later that he was progressing well. An anaerobic gas-forming organism was obtained on cultivation from the muscle removed.—H. D.

Case 13.—Pte. B., wounded September 1, 1916, admitted the same day. Shell fragment. Through and through wound of thigh, the entrance being on the inner side two inches below Poupart's ligament and the exit at a corresponding point on the outer side. Under an anæsthetic the skin wounds were excised and the track syringed with eusol and drained. Fifteen hours later the patient complained of great pain in his thigh and of great thirst. Temperature 101° F., pulse 110. On examination the wound was found to be tender, crepitant, and gassy. A further anæsthetic was given and the whole tract of the missile laid open; the sartorius, rectus femoris and inner edge of vastus externus were found to be in the "black death" stage of gas gangrene. An incision was made from the anterior superior iliac spine to the knee and another along the inner side of the thigh; these were stitched back to healthy skin so as to fully expose the wound. All three muscles showed the "black death" stage of gangrene at the site of the wound, toning down through the "red death" stage to normal muscle as the muscles were traced

down the thigh. The muscular part of the rectus femoris was removed and also the sartorius from its origin to just above the knee. A strip of the vastus externus for nearly its entire length and for a width of three inches was also excised. No other muscles appeared to be affected ; the wound was dressed with gauze soaked in peroxide and afterwards exposed to the sun and air. For three days he was very ill with a rapid feeble pulse, constant hiccough and persistent vomiting ; he however slowly improved, and on September 5, 1916, his pulse was 88 and temperature 100° F. ; the hiccough still persisted. Two days later a part of the skin flap sloughed, after which he improved steadily and was evacuated on September 11, 1916, with a healthy granulating wound which was skin grafted at the Base hospital. The diagnosis of gas gangrene was confirmed by the mobile laboratory.—G. E. N.

Case 14.—Cpl. V., wounded in the left thigh by a shell fragment at 5.30 p.m. on September 19, 1916 : admitted into the station five hours later. General condition was then good ; pulse 100, temperature 99° F. There was an entry wound the size of half-a-crown on the inner and posterior aspect of the thigh at the junction of the middle and upper thirds. The exit wound was at the outer side of the thigh in the middle third—it was very large, admitting the whole hand and the muscles were greatly lacerated. He was operated on immediately after admission ; the missile had passed through the vastus externus and biceps muscles close to the femur and had partially divided the sciatic nerve. The wound was freely drained after irrigation with eusol. He continued to do well for two days, when the evening pulse rose from 104 to 132 and the temperature to 102° F. The skin over the exit wound was now a dirty brown colour and the area in the neighbourhood was swollen and tender ; there was no subcutaneous crackling. The skin discoloration extended to the popliteal space. A further anæsthetic was given and the large exit wound was freely laid open. In the lower part of the wound the vastus externus muscle was found to be gangrenous ; it was non-contractile and gaseous. The infected portion of the muscle was resected until healthy muscle was exposed. In the upper part of the wound the vastus externus was pale and contracted feebly ; with clean instruments a piece was removed for examination and the skin was laid freely open over the muscle. Eusol dressings were applied. Immediately improvement followed and he was evacuated to the base on September 26, 1916, with a normal temperature and pulse. Anaerobic gas-forming organisms were cultivated from the dead muscle and were also obtained on culture from the portion of muscle removed from the upper part of the wound.

Note.—This case shows the immediate improvement after resection of the infecting focus, and also that muscle may look healthy and be contractile and yet be infected.—H. D.

Reviews.

MANUAL OF MEDICINE. By Thomas Kirkpatrick Monro, M.A., M.D. Fourth Edition. London: Baillière, Tindall and Cox. 1917. Pp. xxiv and 1045. $8\frac{1}{4} \times 5\frac{1}{4}$.

It is difficult for anyone who is not engaged in teaching the elements of medicine, and whose student days are remote, to form any proper idea of what is and what is not a good book for the particular purpose. We know that there are certain classics—Sir Thomas Watson, Fagge, Osler, and a multitude of other books which do not aspire, either by aim or execution, to take so high a place. Most of these humbler volumes fulfil the object with which they are written.

When a text-book written by a distinguished namesake of a very distinguished predecessor, published in their usual style by a well-known firm, reaches its fourth edition, it seems there is little more that need be said about it.

R. J. S. S.

HINTS FOR R.A.M.C. OFFICERS. By Ramcorps. Edinburgh: William Bryce. 1918. Pp. 56; $4\frac{1}{2} \times 5\frac{1}{2}$. Price 1s. net.

A little book containing much knowledge really useful to the junior R.A.M.C. officer. Careful assimilation of the facts contained therein would save much valuable time, both for the junior officer and his senior. Perhaps a few hints on the relationship between newly joined M.O.s with regimental and other officers, might have been added with advantage. In Chapter II room for a word of warning on the marking of the Sick Report "Duty," with its sometime unpleasing results to the thoughtless, or cocksure M.O., might have been found.

H. S. D.

OXFORD WAR PRIMERS: BURNS AND THEIR TREATMENT. By Dr. J. M. H. Macleod. London: Oxford University Press. 1918. Pp. viii and 160. $6\frac{1}{2} \times 3\frac{7}{8}$. 6s. net.

The profession will gladly welcome Dr. McLeod's book on "Burns and their Treatment." One notes that the old classification of Dupuytren is adhered to, which perhaps is as well. The new methods of treatment by ambrine and scabbing are most carefully described and well worthy of study. The causation of burns is an exhaustive one and includes practically every known agent except perhaps mustard-seed gas. We are pleased to see the picric acid treatment is still recommended, as it has stood the test of many years. The chapter on Electrical Burns is full of interesting facts. The letterpress is a credit to the publisher and the illustrations are excellent.

INJURIES OF THE FACE AND JAW AND THEIR REPAIR, AND THE TREATMENT OF FRACTURED JAWS. By P. Martinier and Dr. G. Lemerle. Translated by H. Lawson Whale, M.D., F.R.C.S. London: Baillière, Tindall and Cox. 1917. Pp. xx + 345. $6\frac{3}{4} \times 4\frac{3}{8}$. Price 5s. net.

This little book, written by P. Martinier and Dr. G. Lemerle, Professors of the Dental School of Paris, and translated by H. Lawson Whale, M.D., F.R.C.S., Captain, R.A.M.C (T.), describes very efficiently the method of construction of those prosthetic appliances necessary for the repair of facial injuries, and is a mine of mechanical detail regarding these appliances. Particularly useful information is to be found in the chapter on laryngeal prosthesis.

Those wishing to study the treatment of facial injuries caused by gunshot wounds should read this valuable little book carefully. Notwithstanding that it

was originally published in France in the early days of the War, it deals principally with the treatment of injuries due to pathological causes, and very little mention is made of the subject of plastic surgery and bone-graft operations, in which such great progress has been made and such experience gained by the opportunities which only a War such as the present could afford.

In those cases of extensive loss of the mandible or maxilla, for example, it is contended that immediate replacement of the loss should be made by prosthetic appliance. This, although favourable in the cases dealt with in this work, has not proved practicable, on account of the degree of sepsis always present, in the treatment of gunshot wounds.

As to the question of the use of paraffin wax for the restoration of contour, we quite agree with the authors on this point, for experience has proved this to be a failure.

With regard to the description contained in this volume of rhinoplasty over a metal apparatus, this method, in the majority of cases, has only met with unsatisfactory results. The more successful method, and one giving far better results, is that of using cartilage supports; and we agree with the translator in his remarks that the authors' comments on total rhinoplasty seem unwarrantably severe.

On the question of fractures of the lower jaw, the author emphasizes in a marked manner the necessity for the preservation of dental adjustment, in opposition to the other school which advocates approximation of fragments in order to obtain, at the expense of articulation if necessary, bony union. Whilst we favour the latter view, it would seem that the authors have been greatly influenced by the nature of cases met with in civilian practice, where loss of substance, except from surgical interference, is very rarely met with—consequently little or no necessity to obtain bony union.

On p. 282, the authors state that a tooth, although tottering, should never be extracted when it is in the immediate region of the fracture. As the translator states, this point has been much debated; and we feel sure that had the writers of this useful little book had extensive experience with gunshot wounds they would be of our opinion that more bone is lost through septic absorption by the retention of teeth than by their extraction in an early stage.

The outstanding value of the work is in the wonderful ingenuity of the mechanical appliances described, and therefore it should be carefully read by, and in the hands of, all interested in this particular branch of surgery.

H. D. G.

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JOURNAL

OF THE

ROYAL ARMY MEDICAL CORPS.

Corps News.

JUNE, 1918.

EXTRACTS FROM THE "LONDON GAZETTE."

War Office,
April 25, 1918.

His Majesty the King has been graciously pleased to approve of the award of the Military Medal for Bravery in the Field to the undermentioned Non-commissioned Officer and Men :—

ROYAL ARMY MEDICAL CORPS.

No. 403069 Pte. H. Bolton (Leeds).
No. 38122 Pte. A. Coad (Truro).
No. 403424 Pte. J. Edgell (Leeds).
No. 32763 Pte. J. W. Flower (Sheffield).
No. 65201 Pte. F. B. Hands (Edgbaston).
No. 38161 Pte. G. F. Harris (Somers Town).
No. 37479 Pte. W. Jackson (Birmingham).
No. 68445 Pte. J. B. Jones (Merthyr Tydvil).
No. 318033 Serjt. F. J. Johnstone (Glasgow).
No. 37475 Pte. H. S. Lilley (Bolton).
No. 37546 Pte. C. W. Linscott (S. Bermondsey).
No. 92903 Pte. A. J. G. Mackie (Carron).
No. 90768 Pte. H. E. McIlroy (Paddington).
No. 386033 Pte. W. C. Thompson (Newcastle-on-Tyne).

With reference to the awards conferred as announced in the *London Gazette*, dated January 18, 1918, the following are the statements of service for which the decorations were conferred :—

AWARDED THE DISTINGUISHED SERVICE ORDER.

Temp. Capt. Archibald Stirling Kennedy Anderson, M.C., M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. Under heavy shell fire he led forward a party of stretcher-bearers and collected twenty-five wounded men who were lying within fifty yards of the enemy line. He set a splendid example of courage and self-sacrifice.

Temp. Capt. James Thornley Bowman, M.D., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. During an attack he attended to the wounded for five hours in the open under heavy fire. He showed the greatest coolness and courage throughout the day.

Temp. Capt. Howard Boyd Graham, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. During a heavy barrage he personally reconnoitred the advanced aid posts, with the result that many cases were evacuated the moment the barrage lifted. This action undoubtedly saved many lives. On another occasion he went out and brought in two officers and four other ranks who were lying out unattended. Later, hearing that an officer had become a casualty, he went forward and brought him back single-handed to a point whence he was evacuated. His coolness, fearlessness and devotion to duty were beyond praise.

Capt. (Acting Lieut.-Col.) Robert Thin Craig Robertson, M.B., Royal Army Medical Corps (Special Reserve).

For conspicuous gallantry and devotion to duty. When two ammunition lorries were hit by enemy shells he went forward at great risk to where several men had been killed or wounded, dressed their wounds and had them evacuated. He displayed great initiation and coolness.

AWARDED A SECOND BAR TO THE MILITARY CROSS.

Temp. Capt. George D'Rastrik Carr, M.C., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. He reconnoitred a route for his bearers under heavy shelling. From 4 a.m. till late the following evening he was continually under heavy fire between his collecting post and the regimental aid post. His constant knowledge of events in the first line enabled only the requisite number of bearers to be used in the more dangerous parts.

AWARDED A BAR TO THE MILITARY CROSS.

Capt. Robert Bell Stewart, M.C., Royal Army Medical Corps (Special Reserve).

For conspicuous gallantry and devotion to duty. He successfully carried out the evacuation of the wounded from the regimental aid post over extremely bad ground and under very heavy fire.

Temp. Capt. Francis Ruthven Thornton, M.C., M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty when in charge of bearers, evacuating the wounded under very adverse weather conditions. When, owing to an intense barrage, bearers repeatedly failed to get through to the advanced regimental aid post he personally led them forward and remained there for ten hours, evacuating thirty-six serious cases.

AWARDED THE MILITARY CROSS.

Temp. Capt. John Frederick Broughton, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty in attending the wounded in an exposed aid post under heavy fire throughout the day. He visited the advanced positions whenever he could to organize his bearers and collect wounded. After the battalion was relieved he stayed in the line all the following day to search for wounded. When the medical officer of a neighbouring battalion became a casualty he passed from one aid post to the other under heavy fire and attended to the wounded of both battalions.

Capt. Angus Cameron, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. During the attack he handled his company in a most capable manner. When the objective was reached, he collected men of several units, and consolidated the position. It was owing to his prompt action that two bombing attacks were easily repulsed. During the whole operation his courage and cheerfulness were the means of inspiring all ranks.

Temp. Lieut. (Temp. Capt.) William Kealty Campbell, D.S.O., M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. In searching for wounded he and his party came under heavy fire. Ordering his bearers to take cover in shell-holes, he searched the ground by himself, calling up bearers as he required them. When moving away with the last wounded man a shell severely wounded one of the bearers. Ordering the remainder to take their stretcher case into a "pill-box," he remained in the open with the wounded bearer till he died.

Temp. Capt. Charles Launder Chalk, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. This officer displayed great initiative under shell fire. When his dressing station was crowded with wounded he dressed in the open those who were unable to find cover. Through his promptitude and heedlessness of danger in dressing and clearing away the wounded he undoubtedly saved many lives during a critical period.

Capt. Joseph Wilfred Craven, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty when in charge of bearer parties removing the wounded. Discovering many wounded in an old enemy "pill-box," he returned for bearers, and so saved the lives of one officer and six men. During the whole time he was under severe machine-gun fire and shrapnel.

Temp. Capt. Henzell Howard Dummere, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. He voluntarily left shelter and went across difficult ground under very heavy fire to help three seriously wounded men, afterwards conducting the stretcher-bearers to the aid post a mile away.

Temp. Capt. John Tryweryn Lloyd, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty as battalion medical officer. He frequently went forward under heavy machine-gun and shell fire to supervise the work of his stretcher-bearers.

Temp. Capt. James MacGregor, M.D., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty when in charge of a bearer division. He followed up the barrage closely and evacuated the wounded under heavy fire. By magnificent work he cleared the ground of wounded before dusk.

Temp. Capt. James Murdoch MacKay, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. He remained at work although suffering from gas-poisoning, and continually left the aid post to direct his orderlies and bring in walking wounded cases.

Temp. Capt. David Matthew, M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty during two days' heavy fighting. He established a dressing station in an advanced position whence he continually went forward to dress wounded and organize carrying parties under heavy fire. He inspired his men to continue their work by his splendid example.

Capt Alfred James Angel McCabe-Dallas, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. Seeing a trench some distance off being heavily shelled, although not in medical charge of the troops occupying it he at once ran to see if he could help any casualties, and while doing so was severely wounded.

Capt. Philip McRitchie, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. Coming upon a party of sixteen men wounded by shell fire, he dressed their wounds under a heavy barrage and called for volunteers

to carry them to the aid post. Hearing that his Commanding Officer and Adjutant had been wounded about a mile away, he made his way across country under heavy fire and got them to the dressing station.

Capt. Edgar Percival, D.S.O., M.B., Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty when in command of stretcher-bearers. While he was leading a party of sixteen towards the aid post they came under heavy shell fire, and seven were wounded. He got them back and brought up fresh men. One of this party was wounded, and, while an attempt was being made to get him back the patient and three bearers were killed. After replacing these casualties he succeeded in getting through at the third attempt, and removed all the wounded.

Temp. Capt. Maurice Aloysius Power, M.B., R.A.M.C.

For conspicuous gallantry and devotion to duty when in charge of a bearer division. He collected and dressed wounded under heavy fire. On the way to the aid post his bearer was killed by a shell and he was wounded, but he succeeded in clearing his sector of wounded before dark, and then had to be assisted in an exhausted condition to the dressing station.

Temp. Capt. Reginald Thompson Raine, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. During an intense barrage he went out in the open to attend to wounded, and when his stretcher-bearers became casualties he himself carried cases in. Returning later he also brought in the stretcher-bearers, though exposed all the while to an intense shell fire. He showed a magnificent example to all, and undoubtedly saved many lives.

Capt. Christopher Rogers, M.B., R.A.M.C.

For conspicuous gallantry and devotion to duty as medical officer to his battalion during an attack. When his aid post was blown in, and all his staff killed or wounded, he remained at his post, working in the open under continuous shell fire over twenty hours, 200 cases passing through his hands. After most of his stretcher-bearers had been killed or wounded, he brought in a large number of wounded himself until he was exhausted.

Capt. John Gray Ronaldson, M.B., Royal Army Medical Corps, Special Reserve.

For conspicuous gallantry and devotion to duty while in charge of bearers for nine days, almost without rest. He several times went forward under heavy shell fire to attend to gunners who were wounded and lying in the open. During a heavy gas bombardment, in which there were many casualties, he was himself gassed.

Temp. Capt. Reginald Herman Tribe, Royal Army Medical Corps.

For conspicuous gallantry and devotion to duty. Having organized an aid post behind the front line, he dealt untiringly with many wounded cases belonging to several different units, his courage and efforts being the means of saving many lives and much suffering. He showed splendid determination and resource.

COMMUNIQUÉ.

N.B.—This List is for publication in the Press only, and will not be gazetted.

War Office,
May 1, 1918.

The undermentioned is added to the names of those who have been brought to the notice of the Secretary of State for War for valuable services rendered in connection with the War, published on March 13, 1918 :—

Capt. (Temp. Major) O. Challis, Royal Army Medical Corps.

CORRIGENDA.

For Qmr. and Hon. Lieut. Jones, Royal Army Medical Corps (T.F.), *read* Qmr. and Hon. Lieut. T. Jones (extra-regimentally employed).

The following names should read as now stated and not as previously published :—

No. 26813 Serjt. (Acting Staff Serjt.) F. Wills, Royal Army Medical Corps.

Jamacia.

No. 2242 Cpl. J. Thomson, Royal Army Medical Corps.

Malta.

No. 19867 Pte. (Acting Serjt.) W. S. Hughes, Royal Army Medical Corps.

List dated September 18, 1917.

Temp. Lieut.-Col. (Hon. Surg-Col.) W. R. Smith, M.D., Royal Army Medical Corps (T.F.).

For Temp. Major J. G. Taylor, M.B., F.R.C.S., Royal Army Medical Corps, *read* Temp. Major G. Taylor, M.B., F.R.C.S., Royal Army Medical Corps.

ARMY MEDICAL SERVICE.

Col. Samuel G. Moores, C.B., C.M.G., to be Temporary Major-Gen. whilst employed as Director of Medical Services of an Army, dated April 8, 1918.

Major-Gen. William George Birrell, M.B., retires on retired pay, dated May 30, 1918.

Lieut.-Gen. Sir A. T. Sloggett, K.C.B., K.C.M.G., K.C.V.O., F.R.C.S., is placed on retired pay on completion of the term of his appointment as Director-General, dated June 1, 1918.

Major-Gen. (Temp. Lieut.-Gen.) T. H. J. C. Goodwin, C.B., C.M.G., D.S.O., to be Director-General, and to be Lieut.-Gen., dated June 1, 1918.

Col. Herbert Innes Pocock, C.M.G., is placed on retired pay, dated June 2, 1918.

Capt. George H. Stack, M.B., to be Acting Lieut.-Col. whilst in command of a Medical unit, dated January 8, 1918.

Capt. Herbert W. Carson, M.B., relinquishes the acting rank of Lieut.-Col. and reverts to the acting rank of Major (with pay and allowances of his substantive rank), dated November 23, 1917.

ROYAL ARMY MEDICAL CORPS.

Lieut.-Col. Patrick H. Henderson, D.S.O., M.B., to be Temporary Colonel whilst employed as Assistant Director of Medical Services of a Division, dated December 27, 1917.

The undermentioned are appointed Hon. Physicians to the King :—

Dated December 26, 1917.—Lieut.-Col. L. W. Harrison, D.S.O., M.B., Royal Army Medical Corps, *vice* Col. (Hon. Surg.-Gen.) R. S. F. Henderson, C.B., M.D., retired pay ; Col. E. Eckersley, M.B., Army Medical Service, *vice* Surg.-Gen. T. M. Corker, C.B., M.D., retired pay.

Dated January 27, 1918.—Col. S. Macdonald, C.M.G., M.B., Army Medical Service, *vice* Surg.-Gen. Sir W. G. Macpherson, K.C.M.G., C.B., M.B., retired pay.

The undermentioned are appointed Hon. Surgeons to the King :—

Dated December 26, 1917.—Major-Gen. (Temp. Lieut-Gen.) T. H. J. C. Goodwin, C.B., C.M.G., D.S.O., Army Medical Service, *vice* Col. (Hon. Surg.-Gen.) S. Hickson, C.B., M.B., retired pay ; Col. E. M. Pilcher, D.S.O., M.B., F.R.C.S., Army Medical Service, *vice* Col. W. H. Horrocks, C.B., M.B., retired pay.

Major Frederick D. G. Howell, D.S.O., M.C., to be Temporary Lieutenant-Colonel whilst employed as Assistant Director of Medical Services of an Army, dated March 17, 1918 ; Major George E. Ferguson to be Acting Lieutenant-Colonel whilst in command of a Medical Unit, dated March 31, 1918.

The undermentioned to be Acting Lieutenant-Colonels whilst specially employed, date March 29, 1918 :—

Major Thomas J. Potter.
Temp. Capt. (Temp. Major) Frederick G. W. Dean, F.R.C.S.Edin.
Temp. Major (Capt. R.A.M.C., T.F.) Lewis C. Bruce, M.C., M.D., F.R.C.P.
Temp. Major (Capt., R.A.M.C., T.F.) Charles S. Young.
Temp. Capt. Patrick Steele, M.D.

Temp. Hon. Major William J. Richard, M.B., to be Temporary Hon. Lieutenant-Colonel whilst employed at the Merryflats War Hospital, dated March 29, 1918.

Temp. Capt. William Kennedy Taylor to be Acting Major, dated February 27, 1918. (Substituted for the notification in the *Gazette* of April 13, 1918.)

The undermentioned to be Acting Majors whilst specially employed :—

Dated March 29, 1918.—Capt. Joseph W. Houston, D.S.O., M.B., Lieut. (Temp. Capt.) Oliver B. Pratt.

The undermentioned to be Temporary Captains whilst specially employed, dated March 29, 1918 :—

James Taylor, F.R.C.S.Edin.
Arthur J. H. Boyton.
Robert Edwards, M.B.
Hugh F. Sheldon.
George Marshall, M.B.
James O'D. Egan.
William F. Law, M.D., F.R.C.S.I.
Douglas J. Glen, M.B.
John P. Duncan, M.B.
William M. Buchanan, M.B.
Duncan Fletcher.
Peter H. Macdonald, M.B.
James W. Bennett.
David W. Torrance, M.D.
James Philp.
John Porter, M.B.
Adolphe Abrahams, M.D.
John T. McCullagh, M.B.
William E. P. Phillips.
Alexander C. E. Gray, M.D.
Archibald H. M. Robertson, M.B.
Robert M. Fenn, M.B.
Denis F. O'Kelly.
John E. Power.
(Lieut.) Donald Burrows.

The undermentioned Temporary Captain to be Acting Lieutenant-Colonel :—

Dated March 29, 1918 :—(Acting Major) Henry Moore, M.C., whilst in command of a Medical Unit ; Charles J. West, M.D., whilst specially employed.

The undermentioned Temporary Captains relinquish the rank of Acting Major on re-posting :—

Dated April 5, 1918.—John Watson, M.B.

Dated April 9, 1918.—Charles F. Drew, M.B. ; Everard C. Abraham, M.C., M.B.

The undermentioned Captains to be Acting Majors, dated January 4, 1918 :—

Vincent T. Carruthers, M.B., F.R.C.S.Edin.
Leopold A. A. Andrews.
Herbert F. Joynt, M.B.
Owen R. McEwen.
Arthur S. Cane, M.D.

Dated February 8, 1918.—David C. Monro, M.B.

Dated March 12, 1918.—James R. Hill, M.B.

Dated March 16, 1918.—Albert Jackson, whilst specially employed.

Dated March 29, 1918.—John I. W. Morris (Reserve of Officers); Herbert G. Winter, M.C.; Robert M. Dickson, M.D.

The undermentioned Temporary Captains to be Acting Majors, dated January 4, 1918:—

George B. Holroyde.
Wilfrid V. Macaskie, M.B.
Harold B. Atlee, M.C., M.B.
Kenneth G. Fraser.
William K. McIntyre, M.C., M.B.
William H. Peacock, M.B.
William H. Welsh, M.B.
Charles G. Sutherland, M.B.
James P. Good, M.D.
Alan R. Green, M.B.
Francis C. Tibbs.
William D. Cruickshank, M.B.
Alexander C. Sturrock, M.D.
Alexander G. Anderson, M.B.
Isaac Jones, M.D.
Edward B. Gunson, M.D.
William T. Hessel, M.B.
William P. Ker.
Richard A. P. Hill, M.D.
James L. Gordon, M.D.
Lionel D. Woods.
Claude A. H. Gee, M.B.
Robert S. Dewar, M.B.
George V. Bakewell, M.B.
Lancelot G. McCune, M.B.
Samuel Campbell, M.B.
David I. Anderson, M.B.
John Alexander, M.B.
(Temp. Lieut.) Walter B. Griffin, F.R.C.S.
(Temp. Lieut.) Norman B. Stewart, M.B.
(Lieut.) John F. Bourke, M.C.
(Lieut.) St. John D. Buxton.
William A. Rogerson.

Dated January 24, 1918.—Alexander Dick, M.B.

Dated February 12, 1918.—Thomas Ferguson, M.B.

Dated February 27, 1918.—George D. Mathewson, M.B.

Dated March 2, 1918.—Johnston Hughston, M.B.; Robert K. Birnie, M.B.

Dated March 3, 1918.—Arthur P. Saint, M.C.

Dated March 12, 1918.—Robert Scott, M.B.

Dated March 15, 1918.—John H. McNicol, M.C., M.B.

Dated March 29, 1918.—William Martin, M.B.; Frederick N. Brown; James A. W. Watts, M.B.; Harry T. Newling; Francis B. Young, M.B.; William A. Wheeldon, M.B.; John D. Duncan, M.D.; Alfred Griffiths, M.B., F.R.C.S.Edin.; (Temp. Lieut.) Richard G. Oram.

Dated April 7, 1918.—Walter Crabtree, M.B.; James R. Craig, M.B.; William M. Badenock, M.B.

Dated April 11, 1918.—John H. Banks.

Dated April 18, 1918.—James A. Conway, M.C., M.D.

Dated April 20, 1918.—Alan G. Cook, M.B.

The undermentioned Temporary Majors to be Temporary Lieutenant-Colonels:—

Dated May 25, 1918.—Sir Robert Armstrong-Jones, Knt., M.D., F.R.C.P., F.R.C.S.; Hamilton C. Marr, M.D.

The undermentioned to be Acting Lieutenant-Colonels whilst in command of a Medical Unit:—

Dated December 16, 1917.—Captain Charles H. Stringer; Captain Gilbert G. Collet, M.B.; Lieut. (Temp. Capt.) Rudolf W. Galloway, M.B.

Dated January 16, 1918.—Captain Leopold T. Poole, M.C., M.B.

Dated April 1, 1918.—Major Samuel B. Smith, D.S.O., M.D.

Temp. Hon. Major Thomas E. K. Stansfield, M.B., to be Temp. Hon. Lieut.-Col., dated May 25, 1918.

Temp. Major Thomas Basil Rhodes, M.B., relinquishes his commission on ceasing to be employed with the N. Staffordshire Infirmary, dated May 20, 1918.

Temp. Capt (Acting Major) Guy W. Parry relinquishes his commission, dated March 28, 1918.

The undermentioned to be Acting Majors:—

Dated January 4, 1918.—Temp. Capt. Donovan B. Pascall, M.B.; Temp. Capt. Norman A. A. Hughes; Temp. Capt. Allan C. Hancock, M.C.; Temp. Capt. Harold J. de Brent, M.C.; Capt. Clive T. Edmunds (from January 4 to 29, 1918).

Dated April 6, 1918.—Temp. Lieut. Duncan C. Ogilvie; Temp. Capt. Robert Tindall, M.C.

Temp. Capt. William Core, M.B., is dismissed the service by sentence of a General Court-Martial, dated March 14, 1918.

The undermentioned Temporary Captains relinquish their commissions:—

Dated February 21, 1918.—Charles D. Goodenough.

Dated May 1, 1918.—William T. Evans, M.B.

Dated May 25, 1918.—William L. Christie, M.D., F.R.C.S.

Temp. Hon. Capt. William R. Carter, M.B., relinquishes his commission on ceasing to be employed with British Red Cross Society in France, dated April 25, 1918.

The undermentioned Lieutenants (Temporary Captains) to be Captains:—

Dated May 10, 1918.—Kenneth A. M. Tomory, M.B.

Dated May 11, 1918.—John S. Sloper, M.B.; Colin Wilson, M.B.; Harry N. Stafford, M.C.; Robert J. Sullivan, M.C., M.D.

Dated May 16, 1918.—Robert A. Anderson, M.B.

Dated May 28, 1918.—Frederick M. Lipscomb; Harry P. Rudolf, M.B.; Douglas N. Macleod, M.B.

BIRTH.

DENNIS.—On February 18, at Toots, Caversham, Reading, the wife of Lieut.-Col. B. R. Dennis, Royal Army Medical Corps, of a son.

EXCHANGES, &c.

The charge for inserting Notices respecting Exchanges in the Royal Army Medical Corps is 5/- for not more than five lines, which should be forwarded by Cheque or P.O.O., with the notice, to Messrs. G. STREET and CO., Ltd., 8, Serle Street, London, W.C., not later than the 22nd of the month.

Notices.

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Foreign: United States Public Health Service, The Military Surgeon, Archives Médicales Belges, Bulletin de l'Institut Pasteur, Bulletin de la Société de Pathologie Exotique, Giornale di Medicina Militare, Archives de Médecine et Pharmacie Navales, Colonies et Marine, Tidskrift I Militär Hälsovard.

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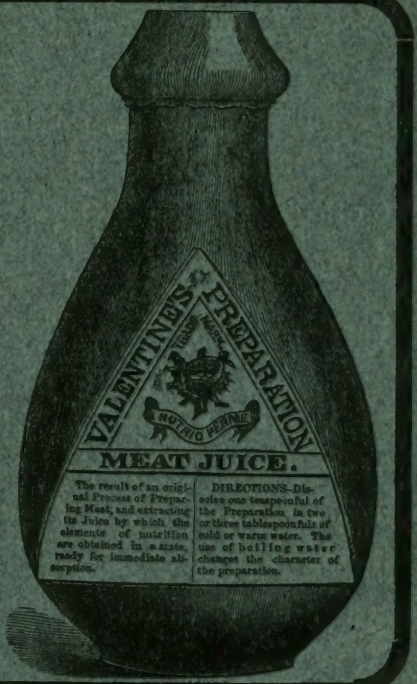
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